EXHIBIT C


PAUL J. MICHAELS, M.D.

BOARD CERTIFIED IN ANATOMIC AND CLINICAL PATHOLOGY, AND CYTOPATHOLOGY


Expert Report of Paul J. Michaels, M.D.
(Re: General Opinions)

BACKGROUND:

I am certified by the American Board of Pathology in Anatomic Pathology, Clinical Pathology, and Cytopathology. I attended and received my medical degree from the University of California, Los Angeles School of Medicine. I completed a residency in anatomic and clinical pathology at Massachusetts General Hospital, an affiliate of the Harvard School of Medicine, where I was a Clinical Fellow in Pathology. Following my residency, I completed a year of subspecialization in Cytopathology, also at Massachusetts General Hospital.

I am a pathologist affiliated with Clinical Pathology Associates in Austin, Texas. I have staff privileges at University Medical Center at Brackenridge, Seton Medical Center Austin, St. David's Medical Center, Seton Northwest Hospital, Seton Southwest Hospital, Seton Highland Lakes Hospital, Dell Children's Medical Center of Central Texas, Arise Austin Medical Center, Westlake Medical Center, Central Texas Medical Center (San Marcos, TX), and Resolute Health Hospital (New Braunfels, TX). Presently, I am the Laboratory Director for two separate Stat clinical laboratories in the Austin area, both affiliated with Clinical Pathology Laboratory/Sonic Healthcare USA, the third largest pathology company in the United States. During my career, I have had a strong subspecialty focus in breast and gynecology pathology, as well as cytopathology. I regularly attend and participate in tumor multidisciplinary conferences. In addition, I was a contracted speaker with Genomic Health, Inc., specializing in the Onco*type* DX Breast Cancer Assay. I was also an invited speaker at the annual National Interdisciplinary Breast Center Conference for the National Consortium of Breast Centers, both in 2012 and 2013, lecturing on various topics in the field of breast cancer. My current curriculum vitae is attached to this report.

I have been asked to provide an expert report regarding my general opinions as they relate to this litigation. In preparation for providing this opinion, I have reviewed numerous studies published in the scientific literature, as well as various Ethicon documents, deposition testimony, and other materials in arriving at my findings and opinions in this matter, a list of which is attached to my report. All of my opinions stated below are held to a reasonable degree of medical and scientific certainty and I reserve the right to modify or change my opinions based on further documents or information that may be provided to me in the future.

COMMENT:

Not long after the commencement of transvaginal mesh (TVM) repair for POP and SUI, many complications were reported as being directly related to sequela from the host response of the implanted synthetic graft. The most common complications included mesh erosion and extrusion of the mesh through the vagina, pain, bleeding, secondary infection, dyspareunia, urinary problems, partner pain, and even organ fistula formation. Many of these complications required repeat surgical intervention. The pathologic response to the synthetic grafts used in surgery depends in large part on the physical and structural properties of the prosthesis. This host response varies based on mesh absorbability, pore size (size between filaments), and overall weight/density.

While absorbable material initially elicits a chronic foreign body inflammatory reaction, following complete absorption and subsequent fibroblast proliferation, the material is replaced by collagen-rich connective tissue, devoid of most acute or chronic inflammatory elements with obvious resolution of any foreign body reaction (Klinge 1999; Klinge 2001). In contrast, non-absorbable prosthetic material such as polypropylene is typically characterized by a persistent inflammatory response with ongoing foreign body-type giant cells (tissue macrophages), chronic inflammatory cells, and neovascularization. A study of modified mesh material used experimentally in a surgical setting that contained a mixture of non-absorbable polypropylene and absorbable polyglactin showed that reducing the amount of polypropylene (non-absorbable) to less than 30%, though still providing the necessary mechanical stability, significantly reduced the degree of inflammation and subsequent fibrosis, leading to increased mesh flexibility (Klinge 1998). In a separate experimental study in dogs, also by Klinge and colleagues (1998), a multifilament combination of nonabsorbable polypropylene and absorbable polyglactin histologically showed considerably less inflammation and stromal fibrosis, compared to monofilament polypropylene grafts.

Studies over many years have uniformly supported the finding that larger mesh pore sizes have better incorporation into the surrounding native tissues (Greca 2001; Klinge 2002; Weyhe 2006). Whereas smaller pore sizes significantly impair vessel and adipose tissue penetration secondary to prominent fibrosis between adjacent mesh filaments (“bridging fibrosis”) (Chvapil 1969; Klinge 1999; Klinge 2002; Klosterhalfen 2005; Cobb 2006), larger-pore mesh material allows for infiltration by vascularized connective fibroadipose tissue (Taylor 1972; Cobb 2015) both structurally allowing for reduced fibrosis with subsequent retention of flexibility (Orenstein 2012; Lake 2015) and decreasing the ability for infection by bacteria introduced into the surgical site at the time of implantation (Merrit 1979). Additionally, the foreign body-type giant cell response and prominent fibrosis invariably encasing small pore meshes often forms a capsule surrounding the whole mesh (“scar plate”), resulting in the mesh becoming stiff, contracted, and nonflexible (Klosterhalfen 2005). Therefore, mesh contraction is defined

by the reduction in surface area of the original implanted graft due to a retraction of the fibrotic scar tissue around the mesh.

Meshes with larger pore size generally have less material per square unit of measurement and are therefore of a lower weight, whereas those with smaller pore sizes are heavier. Analogous to the data showing a favorable host response with large pore size mesh, studies have concluded that light weight synthetic mesh shows better tissue integration with less inflammation and scar fibrosis (Klinge 1999; Klinge 2002; Klosterhalfen 2005), while the extent of stiffness increased directly in relationship to mesh weight (Cobb 2006).

In addition to the above described histological tissue responses and morphological modifications to implanted synthetic graft material, the clinical symptom of pain has become a significant postoperative complication in surgical cases using polypropylene mesh. The etiology of postsurgical pain can obviously be multifactorial, and several authors have evaluated the clinical consequence of pain in the context of patients with mesh grafts. In some instances, the marked mesh contraction secondary to bridging fibrosis and scar plate formation leads to erosion through the vaginal wall with a resulting acute inflammatory response, triggering regional pain.

Inflammation is a complex tissue reaction to injurious agents resulting in vascular responses, migration and activation of leukocytes, and, occasionally, systemic consequences. Inflammation is divided into acute and chronic patterns.

Acute inflammation has three major components including alteration in vascular caliber and blood flow, structural changes in the microvasculature that permit plasma proteins and leukocytes to leave the circulation, and emigration of leukocytes, mainly neutrophils, from the microcirculation to the focus of injury. Acute inflammatory reactions can be triggered by a variety of stimuli, including foreign bodies. Vasodilation is one of the principle manifestations of acute inflammation which results in increased blood flow that often manifests as an area of increased heat and redness. Clinically, these features not infrequently lead to a complaint of pain at the site of increased blood flow.

Classically, the presence of neutrophils is the hallmark of acute inflammation within tissue. Once neutrophils have migrated from the vascular space into the tissue, they become activated leading to the production of arachidonic acid metabolites, degranulation and secretion of lysosomal enzymes, activation of the oxidative burst, and secretion of cytokines, all processes which contribute to regional pain at the site of inflammation/injury (Figure 1).[1] It would be expected that the inherently destructive

[1] Unless otherwise stated, all figures contained herein are microphotographs of explanted Prolene polypropylene-based mesh material analyzed by me in my role as an expert witness on behalf of the plaintiffs and are used solely for the purpose of demonstrating the morphological features of explanted mesh

nature of the acute inflammatory response would need controls to minimize the extent of tissue damage. Generally, the degree of inflammation declines as the mediators of inflammation have short half-lives and are degraded after being released. However, in the case of a persistent stimulus, such as a non-absorbable mesh, in a compromised anatomic region, such as atrophic vaginal mucosal tissue, the ongoing contraction and migration of the foreign body can lead to continued tissue injury with associated clinical sequalae such as pain.

Chronic inflammation is considered to be an inflammatory reaction in which active inflammation, tissue destruction, and attempts at repair are occurring concurrently within a defined region. Like acute inflammation, there are numerous etiologies for chronic inflammation, again including prolonged exposure to potentially toxic endogenous or exogenous agents. An example of such an exogenous agent would be non-absorbable polypropylene mesh. In contrast to acute inflammation, which is manifested by various vascular changes and a predominantly neutrophilic infiltration, chronic inflammation is characterized by involvement by mononuclear cells, mainly lymphocytes and macrophages, and connective tissue replacement of damaged tissue. The macrophage is typically the dominant cell noted in the context of a chronic inflammatory reaction. Macrophages, once activated, like neutrophils, secrete a wide variety of biologically active products. However, in the case of macrophages, this cascade of events leads to the recruitment of other inflammatory cells, namely lymphocytes, which can ultimately result in tissue fibrosis. The bidirectional interaction of lymphocytes and macrophages together is characteristic of chronic inflammatory responses, as macrophages display foreign antigens to T lymphocytes, which ultimately stimulates T-cell responses. The chronic inflammatory response is typically the dominant type of local immune reaction seen in foreign body reactions, such as to synthetic mesh (Figure 2).

Granulomatous inflammation is a distinctive pattern of a chronic inflammatory reaction encountered in a limited number of immunologically mediated, infectious and non-infectious conditions. One type of granuloma, termed a foreign body granuloma, as the name implies develops when foreign material is introduced into the tissue and is large enough to preclude phagocytosis by a single macrophage. This reaction is often characterized by the presence of multinucleated giant cells, representative of fused epithelioid macrophages (Figures 3-5).

The chronic inflammatory response is closely intertwined with the process of repair. During repair, the injured tissue is replaced through regeneration of native parenchymal cells, by filling of the defect with fibrous tissue (scarring) or, most commonly, by a combination of these two processes. Tissue repair by fibrosis, though an attempt at healing with subsequent strengthening and repair of the tissue, may be harmful depending on the degree of collagen deposition and anatomic location in which it occurs.

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specimens which correlate to the pathological response to polypropylene mesh material described in the medical and scientific publications relied on by me in formulating my opinions.

In a broad sense, the term "fibrosis" applies to any abnormal deposition of connective tissue, though the degree of such deposition will determine the functional impairment, if any. When large defects are initially present, or in the case of some larger foreign bodies, a greater degree of granulation tissue is formed and subsequent wound contraction can occur. Fibrotic bridging is a histological phenomenon closely associated with the clinical consequence of mesh shrinkage. Fibrotic bridging refers to collagen deposition and inflammatory cell infiltration exceeding more than half of the pore size of the mesh (Klinge 2002) (Figures 6-11).

Similarly movement and shrinkage/kinking of the mesh may lead to migration towards nearby structures, ultimately causing fistulas, organ dysfunction, or even perforation (Figures 12-15). In contrast, more subtle histological features have also been shown to correlate with an increased sensation of pain. A study by Bendavid et al (2015) showed that mesh explants in patients complaining of pain contained a higher nerve density compared to tissue examined from patients who simply experienced a hernia recurrence. In this study, many of the nerves showed distortion and entrapment by the mesh material and fibrosis, while occasional areas resulted in the microscopic appearance of a marked neural proliferation, termed a "traumatic neuroma" (Figures 16-18). Therefore, in patients experiencing chronic pain and/or dyspareunia, studies showing significant resolution of symptoms following removal of the mesh (Firoozi 2012; Crosby 2014; Danford 2015), support the idea that nerve proliferation and entrapment by fibrosis/scaring between the mesh filaments as a likely etiology.

With respect to the histologic features that accompany these functional properties, an Ethicon scientist, Dr. Joerg Holste (2005) noted that such meshes lead to excessive scar plate formation, while others, including Dr. Klinge and colleagues, found that the degree and quality of the fibrosis was directly related to the amount of the inflammatory reaction and associated foreign body reaction at the interface between the mesh and the patient's tissue. These cellular responses result in subsequent restriction of the graft, leading to significant complications of chronic pain. In addition, it is clear from Ethicon's internal documents that its polypropylene mesh products are associated with considerable mesh contraction resulting from the fibrous stromal reaction in their surgical meshes containing polypropylene (ETH.MESH.01774758). When mesh contracts or shrinks, it can cause the patient to experience complications, including scarring and chronic pain. According to Ethicon's Medical Affairs Director, Piet Hinoul, who testified in 2013 in *Gross, et al vs Gynecare et al,* complications associated with its Prolift device include histological findings of a significantly scarred vagina with life-long risk of erosion, mesh contraction resulting in severe, chronic pain, and the presence of a severe, chronic inflammatory reaction to the mesh material in some patients resulting in the formation of a scar plate and/or bridging fibrosis. According to Ethicon's internal documents there is "significant evidence that the complications associated with synthetic meshes can cause significant morbidity including infection, erosion, exposure, and pain"

In a 2007 presentation prepared by Ethicon's Research and Development (R&D) department, it was concluded that for an ideal vaginal mesh to not ultimately result in a negative sexual impact for the patient, the graft material would ideally be lightweight and with a large pore size (ETH.MESH.01218361-01218367). With respect to the use of this product in the vaginal floor, Ethicon's internal documents demonstrate that "the vaginal tissue to be augmented is often structurally compromised, atrophic, and devascularized. Such poor tissue quality increased the risk of poor tissue incorporation into the mesh potentially resulting in suboptimal healing and mesh exposure or erosion into an adjacent viscous." This was further verified in 2008 by Dr. Klosterhalfen, the Head of the Duren Institute who was hired as an outside pathology consultant for Ethicon, who summarized his microscopic findings in these cases by noting that the "foreign body tissue reaction followed by secondary fibrosis seems to play a special role in pelvic floor repair" (ETH.MESH.00006636). He actually had informed Ethicon two years prior (2006) that, also based on his studies, the foreign body reaction to these meshes can occur for up to 20 years (ETH.MESH.00870466). Additionally in 2008, he went on to state that this inflammatory reaction is important "because soft tissue coverage is thin in pelvic floor repair" and "fibrosis and folding in this area induce mesh erosions and ulcerations." In a following report delivered the next year, Dr. Klosterhalfen reported, following his histological evaluation of an additional 172 prolapse mesh specimens, that "fibrosis inevitably leads to mechanical irritation, particularly when wrinkling occurs, and should be seen as the basic cause of mesh-induced erosion and ulceration," leading to a setting in which "infection is commonly observed following erosion in the vaginal mucosa" (ETH.MESH.02157879-02157880). Ethicon's documents demonstrate that over the course of Dr. Klosterhalfen's interactions and meetings with Ethicon, he made numerous suggestions aimed at improving the biocompatible nature of mesh implants, including with regards to the choice and weight of the material used, the pore size, and the mechanical characteristics of the mesh products.

Finally, there are numerous publications and internal Ethicon documents that have demonstrated that polypropylene, including Ethicon's Prolene used to manufacture its SUI and POP mesh devices, undergoes *in vivo* degradation over time (Liebert 1976; Jongebloed 1986; Mary 1998; Costello 2007; Clave 2010; Wood 2013; ETH.MESH.15955438; ETH.MESH.1595540; ETH.MESH.15955463; ETH.MESH.13334286) (Figure 19). After implantation of polypropylene, the inflammatory response to the foreign body causes an oxidative burst of free radicals and peroxides leading to embrittlement, crack formation, and loss of mechanical properties (Mary 1998). It has also been found that cholesterol and esterified fatty acids can diffuse into the amorphous zones of polypropylene and impact its physical and mechanical properties, causing damage to the surface (Clave 2010). In many cases, within the cracked and degraded surface layer, blue synthetic granules consistent with a blue pigment that Ethicon adds to the polypropylene resin during the manufacturing process to color some of the mesh fibers blue to aid in visibility can be seen. This finding rules out the possibility that the cracked surface layer is biological, a conclusion which was reached by Ethicon's own scientists in 1984 who used polarization light microscopy

(ETH.MESH.15955462). Surface degradation of the polypropylene, including Ethicon's Prolene-based mesh devices, causes the device to become brittle and crack. This phenomenon increases the inflammatory and foreign body reaction and is a contributing cause of the complications experienced by patients (Mary 1998; Clave 2010).

SUMMARY OF OPINIONS:

1. Prolene Polypropylene surgical mesh, including that contained within many of Ethicon's stress urinary incontinence (SUI) and pelvic organ prolapse (POP) mesh devices, elicits a chronic foreign body inflammatory reaction in tissue;
2. Certain design features of polypropylene surgical mesh lead to scar bridging between polypropylene fibers and scar plating with encapsulation;
3. During repair of the tissue damaged by placement of the mesh device, the wound site contracts and shrinks the implant area;
4. Bridging fibrosis, scar plating/encapsulation, and shrinkage lead to a hardened, rigid device that is damaging to the surrounding vaginal mucosa;
5. Ethicon's Prolene-based meshes degrade and crack *in vivo* contributing to the inflammatory and foreign body reaction and associated complications;
6. The foreign body inflammatory reaction and resultant scarring and mesh contraction can lead to mesh-related complications like nerve entrapment (pain), erosion and extrusion, sexual pain, and urinary/bowel dysfunction.

FIGURES

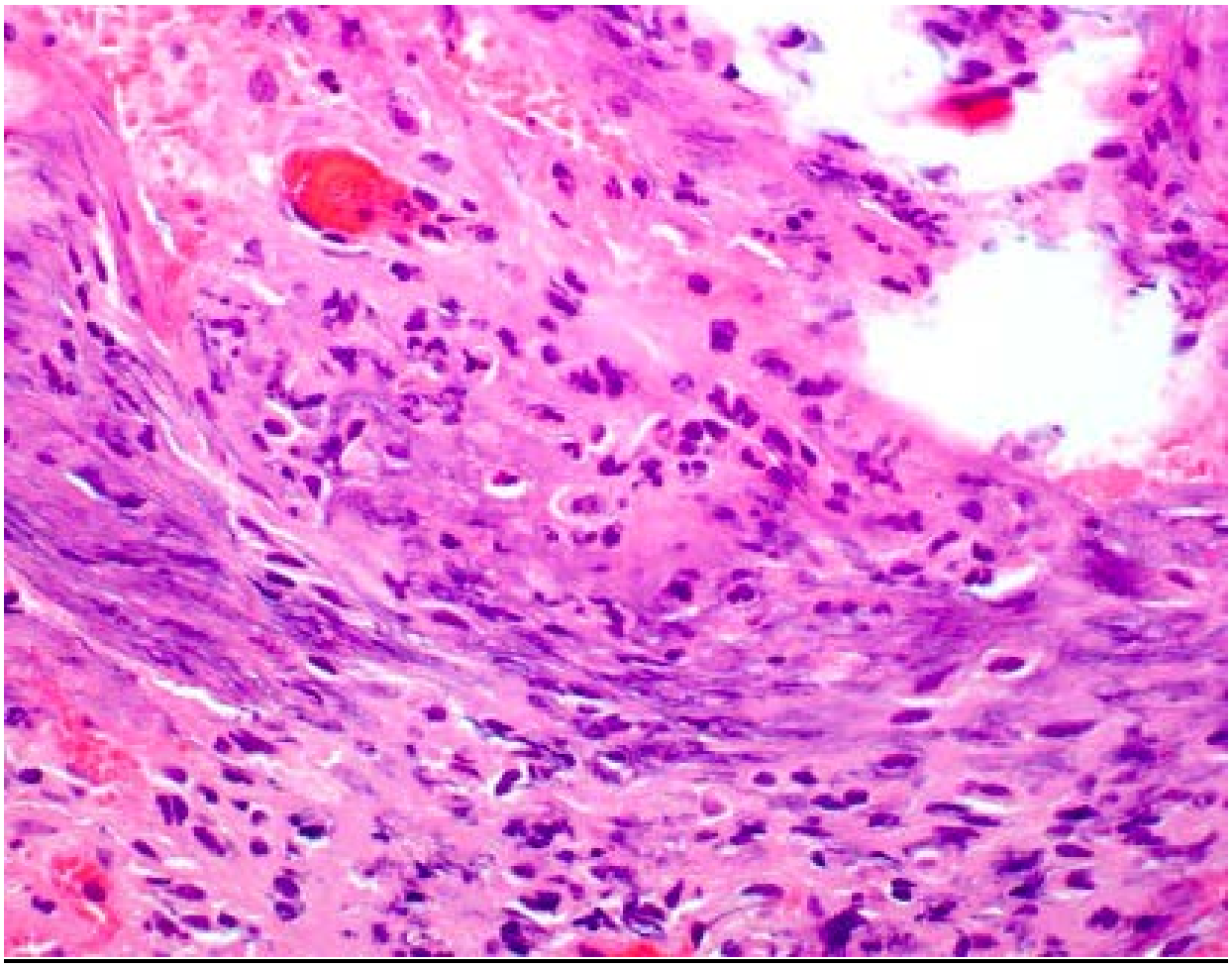

Figure 1: H&E histological specimen explanted from Plaintiff "MM" demonstrating acute inflammation present in areas associated with the mesh and fibrosis, characterized by increased numbers of neutrophils in the stromal tissue (400x magnification).

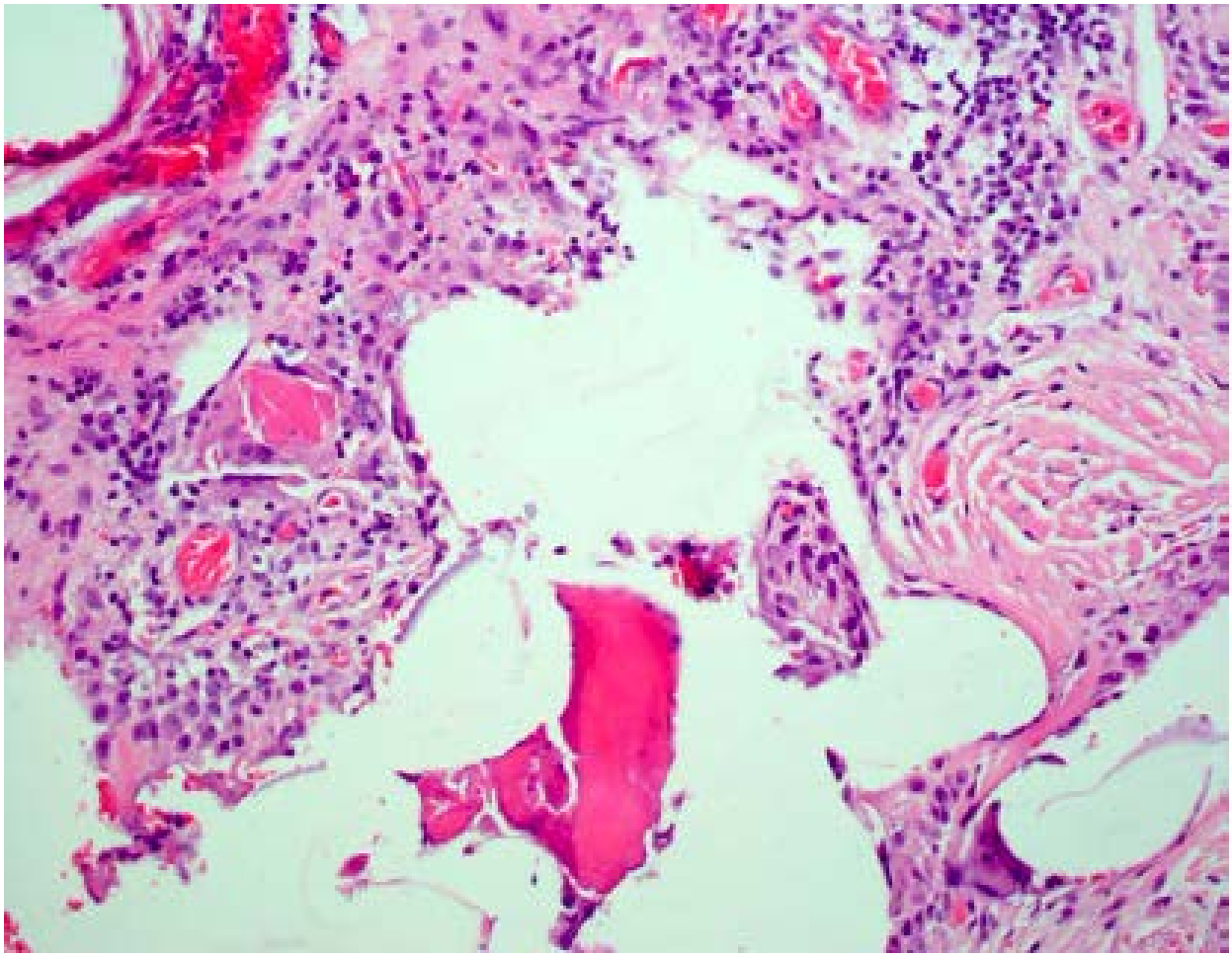

Figure 2: Mesh explanted from Plaintiff "MM" showing mesh filaments surrounded by a prominent chronic inflammatory infiltrate composed predominately by lymphocytes and macrophages with some congested blood vessels (200x magnification).

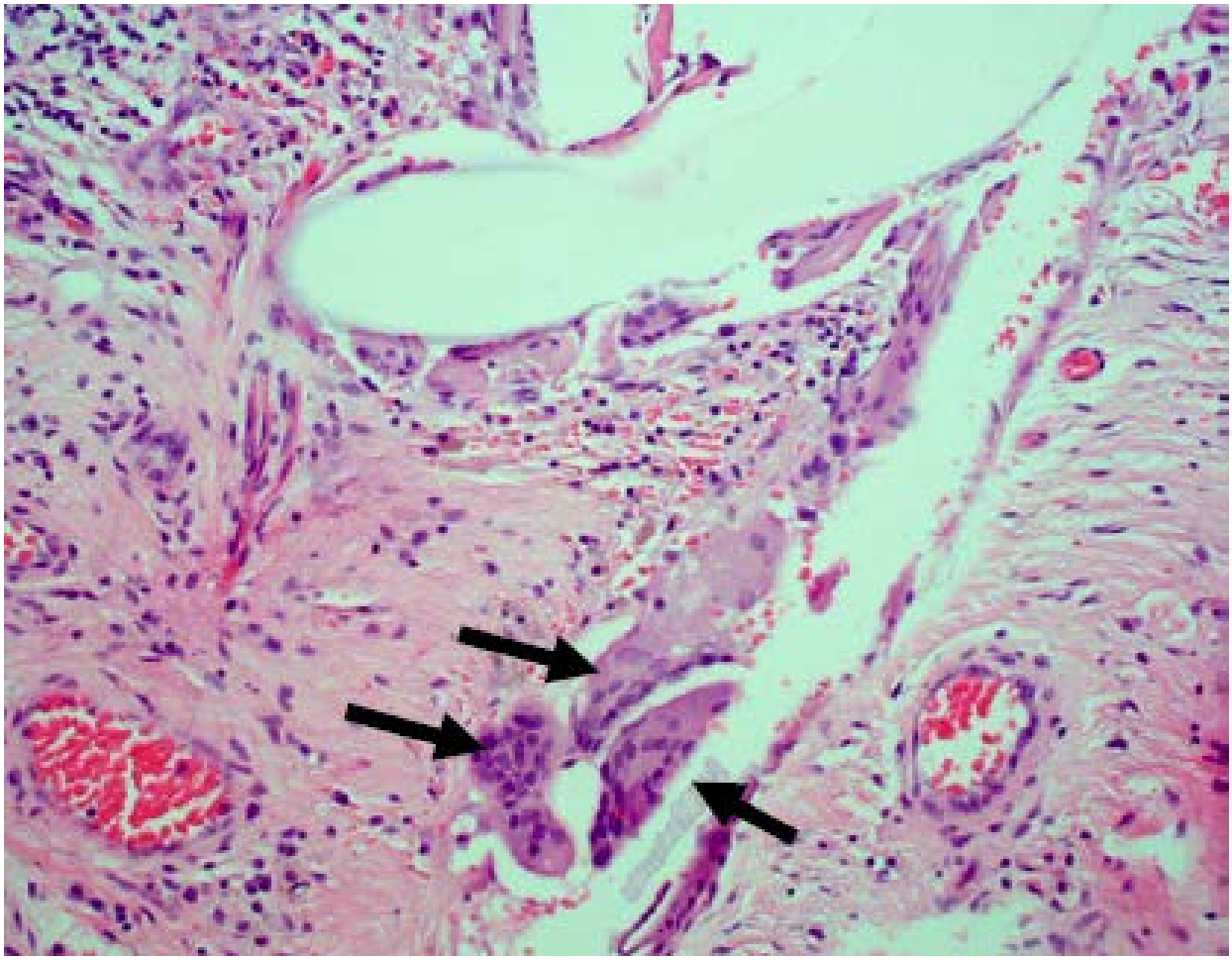

Figure 3: H&E of mesh explanted from Plaintiff "MM" showing vaginal mesh filaments surrounded by numerous, large foreign body multinucleated giant cells (macrophages) with chronic inflammation and dilated blood vessels (200x magnification).

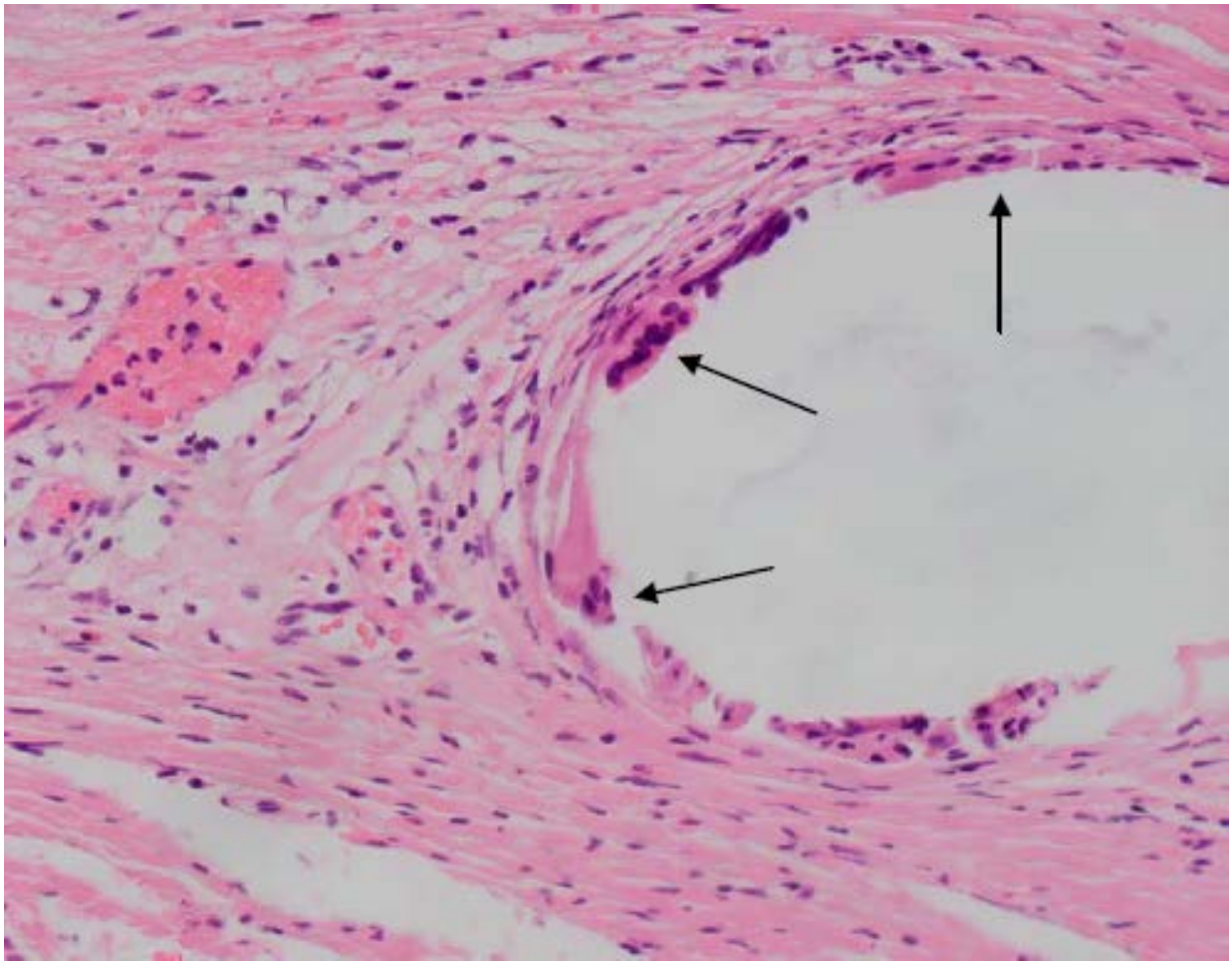

Figure 4: H&E of mesh explanted from Plaintiff "SC" demonstrating foreign body multinucleated giant cells (arrows) surrounded mesh filaments and is accompanied by a chronic inflammatory tissue reaction (400x magnification).

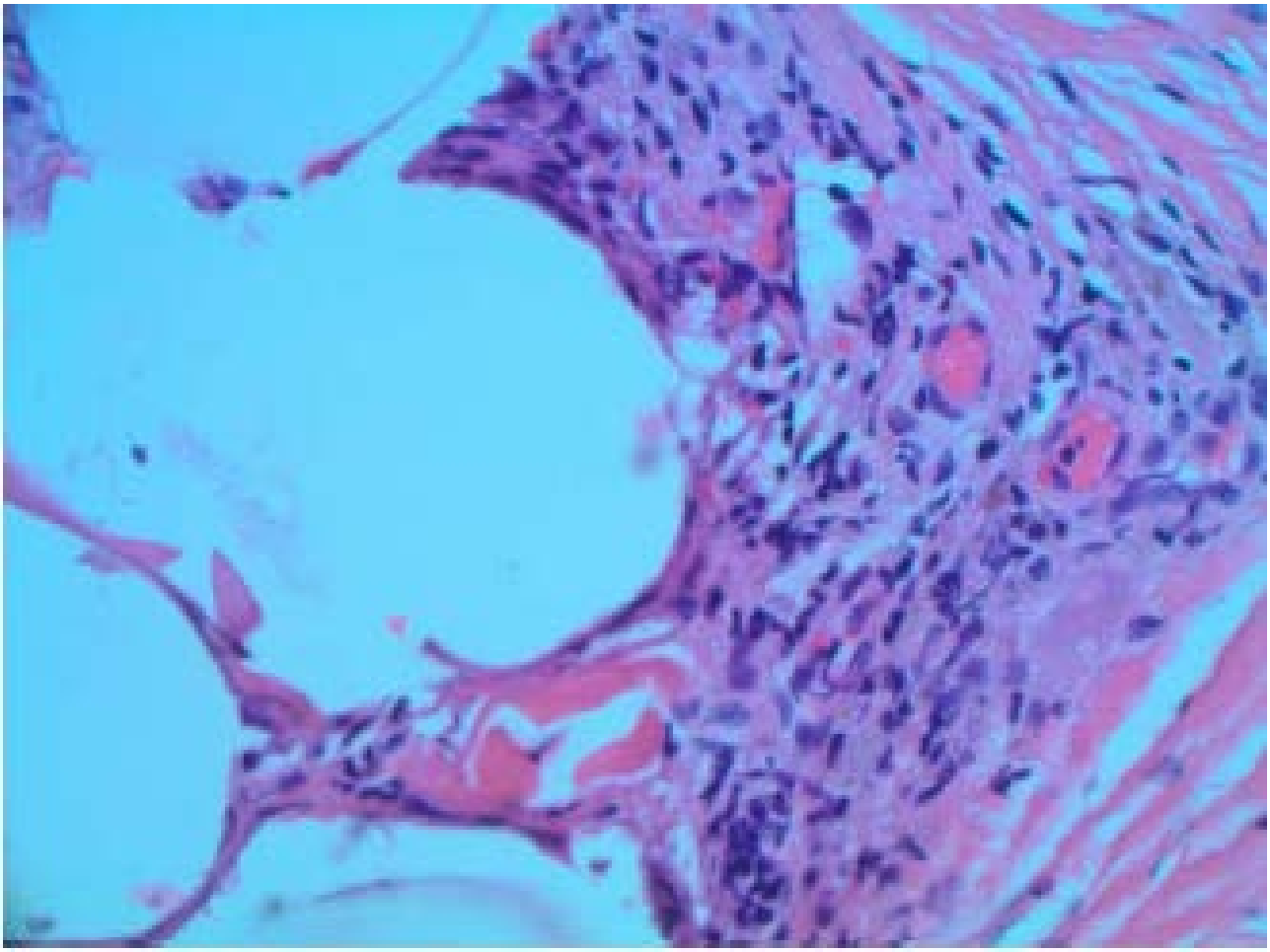

Figure 5: H&E of mesh explanted from Plaintiff "TC" demonstrating granulomatous tissue reaction associated with adjacent mesh filaments (400x magnification).

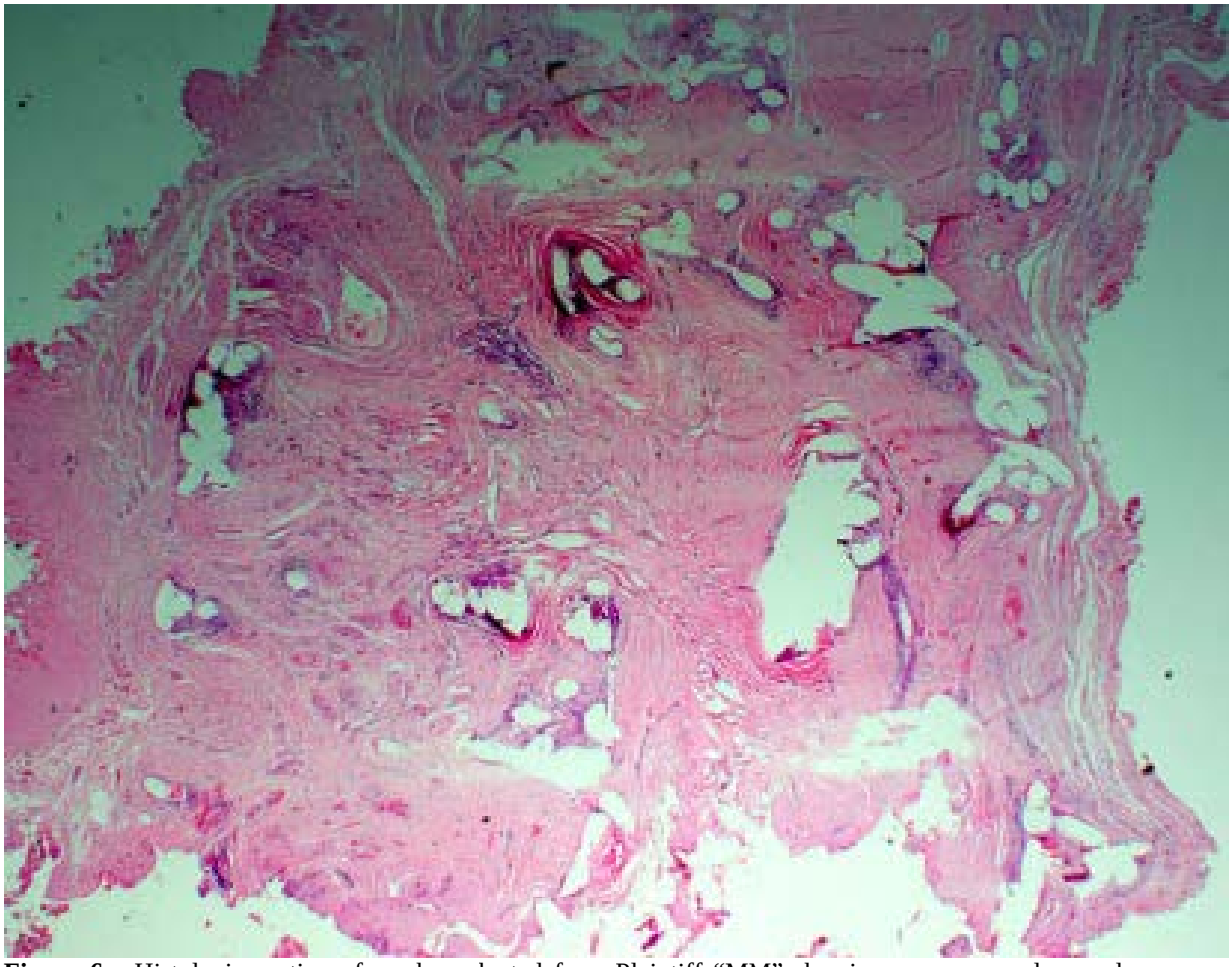

Figure 6: Histologic section of mesh explanted from Plaintiff "MM" showing numerous polypropylene filaments, each surrounded by foreign body granulomas and chronic inflammation, separated by dense areas of fibrosis without intervening adipose tissue ("bridging fibrosis") (40x magnification).

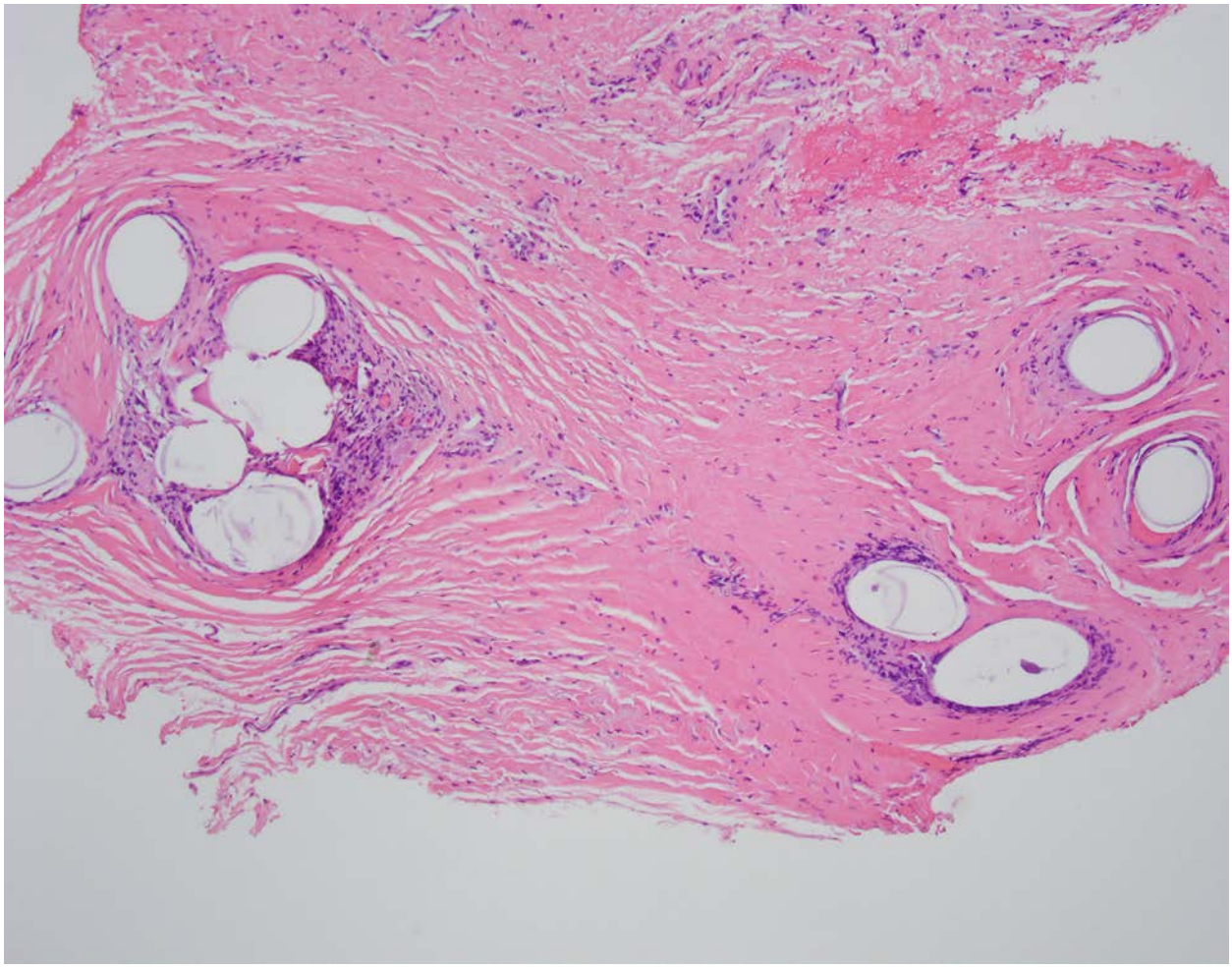

Figure 7: H&E of mesh explanted from Plaintiff "TC" showing bridging fibrosis separating areas of mesh filaments with chronic inflammation (100x magnification).

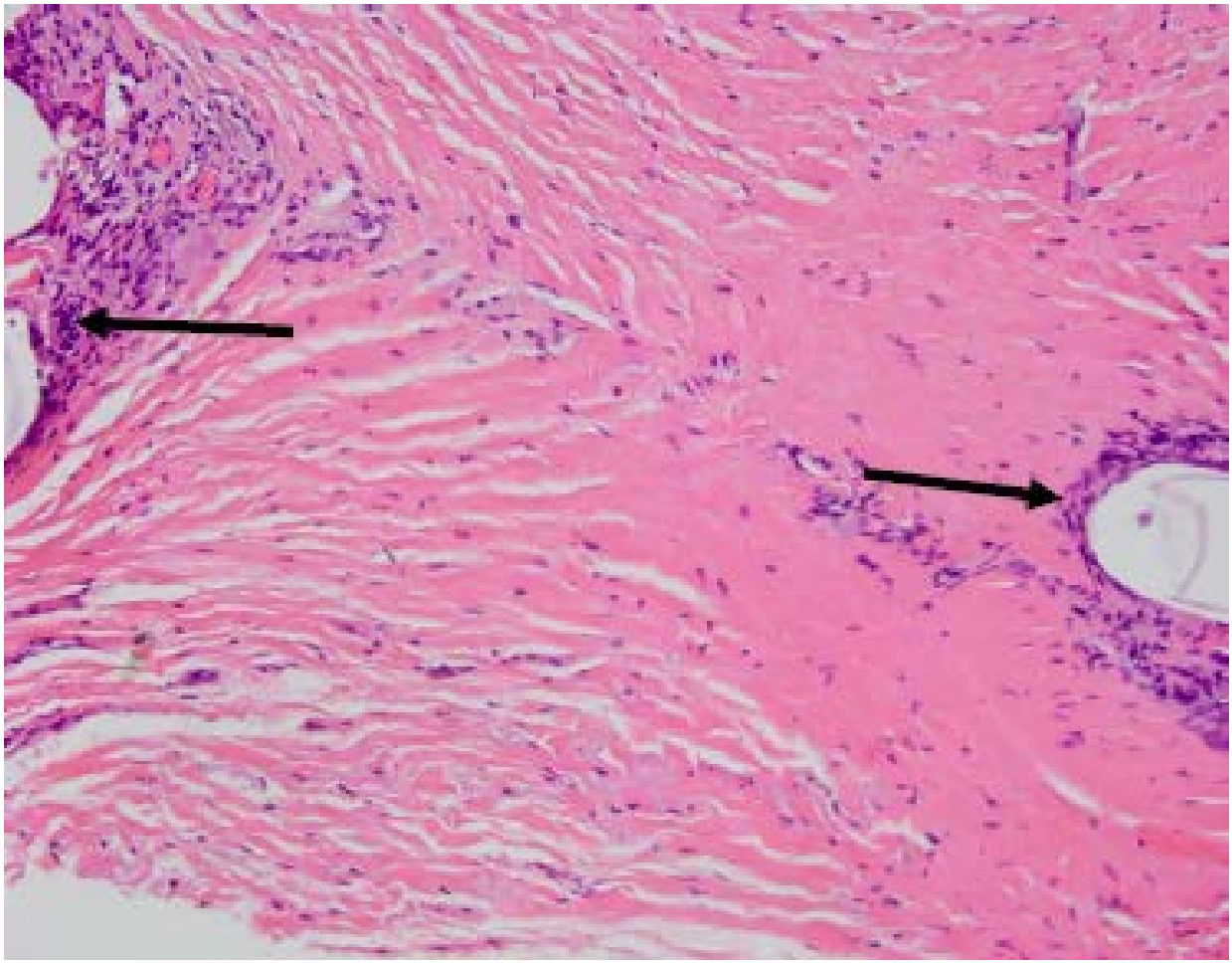

Figure 8: H&E of explanted mesh from Plaintiff "TC" showing prominent hypocellular and hyalinized fibrosis extending between mesh filaments (arrows) (200x magnification).

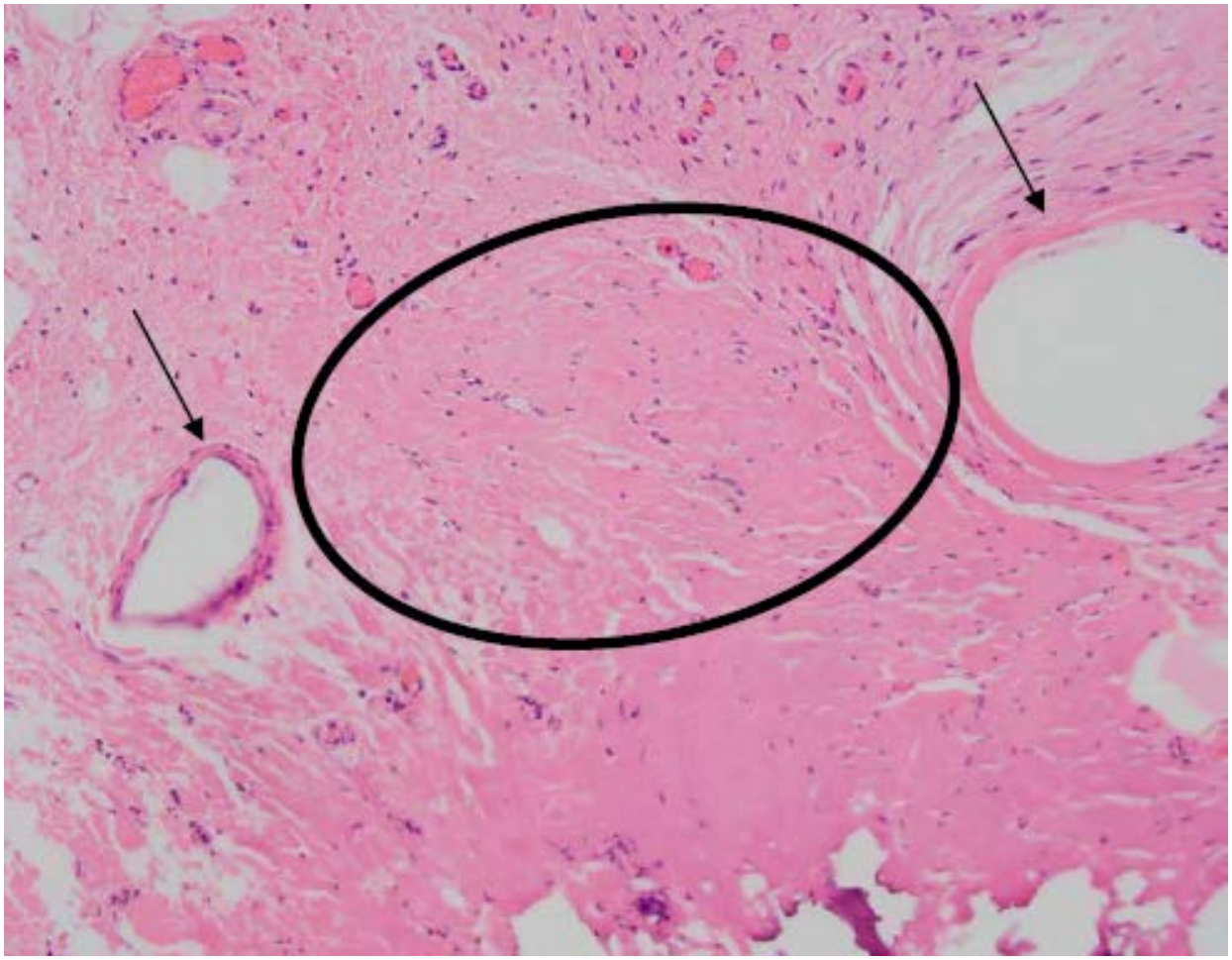

Figure 9: H&E of mesh specimen explanted from Plaintiff "SC" showing area of bridging fibrosis (circle) separating mesh filaments (arrows) (200x magnification).

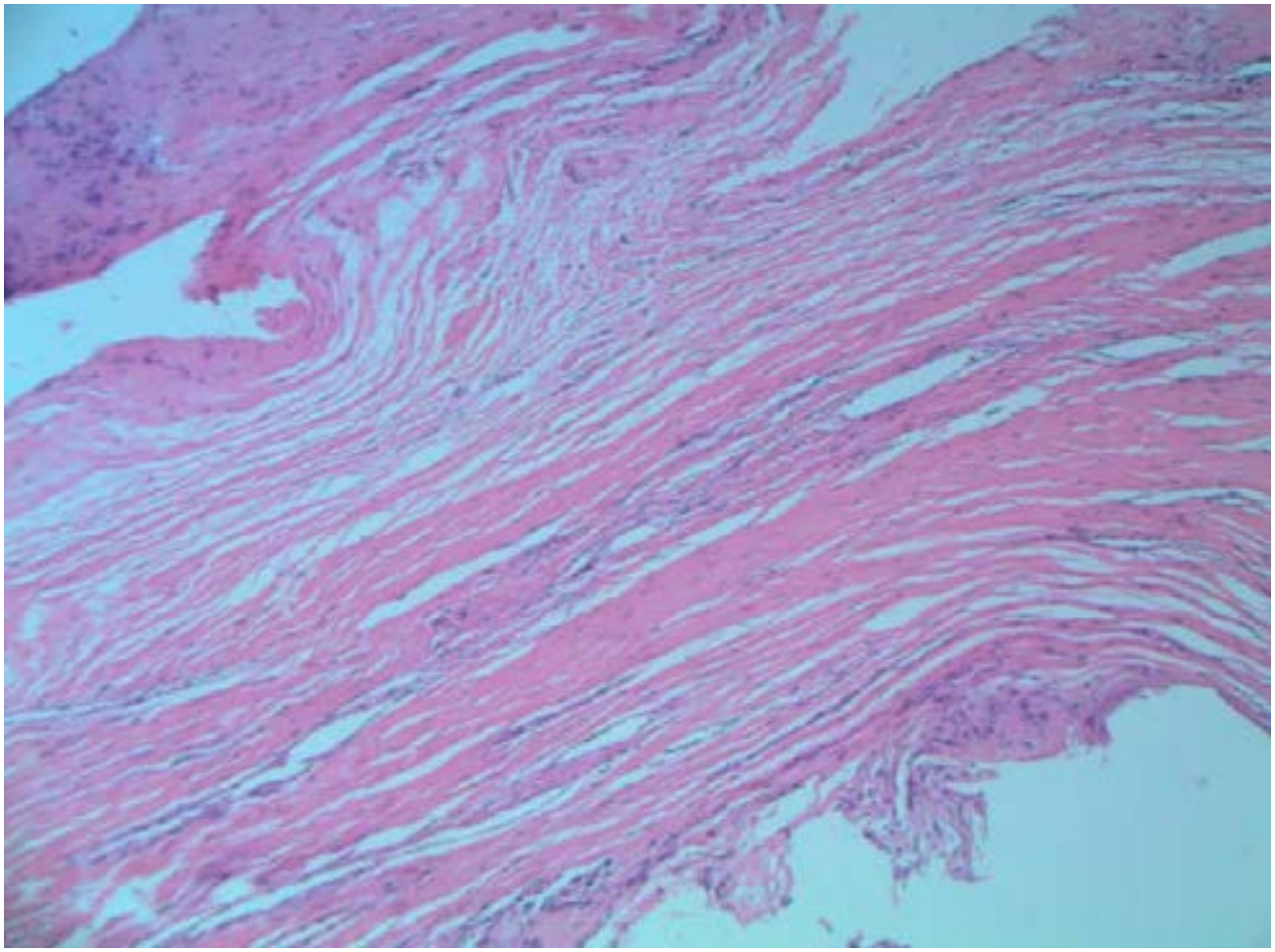

Figure 10: H&E of mesh explanted from Plaintiff "MS" showing bridging fibrosis separating two mesh filament spaces (200x magnification).

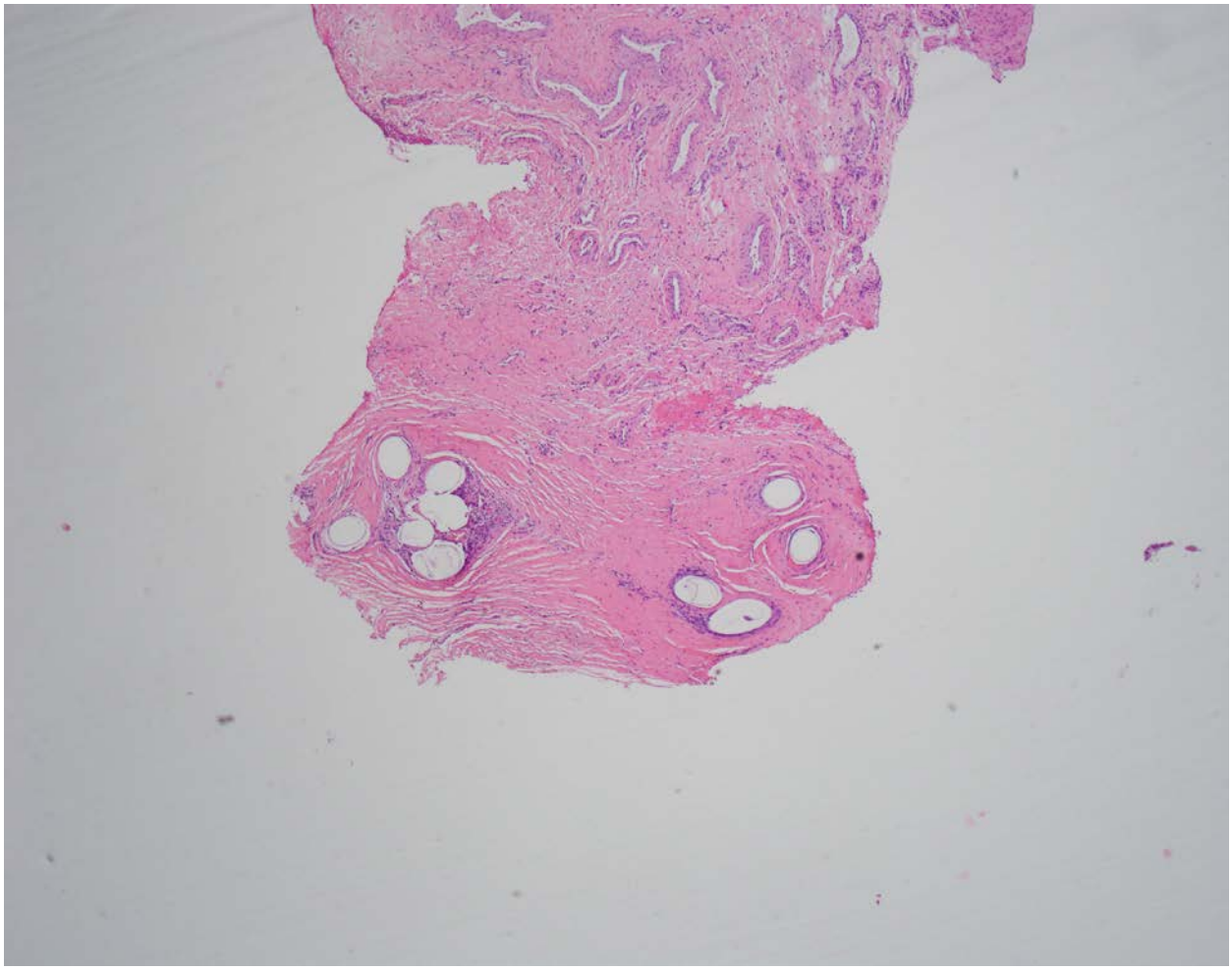

Figure 11: H&E of mesh explanted from Plaintiff "TC" showing encapsulating fibrosis ("scar plate") surrounding mesh filaments (40x magnification).

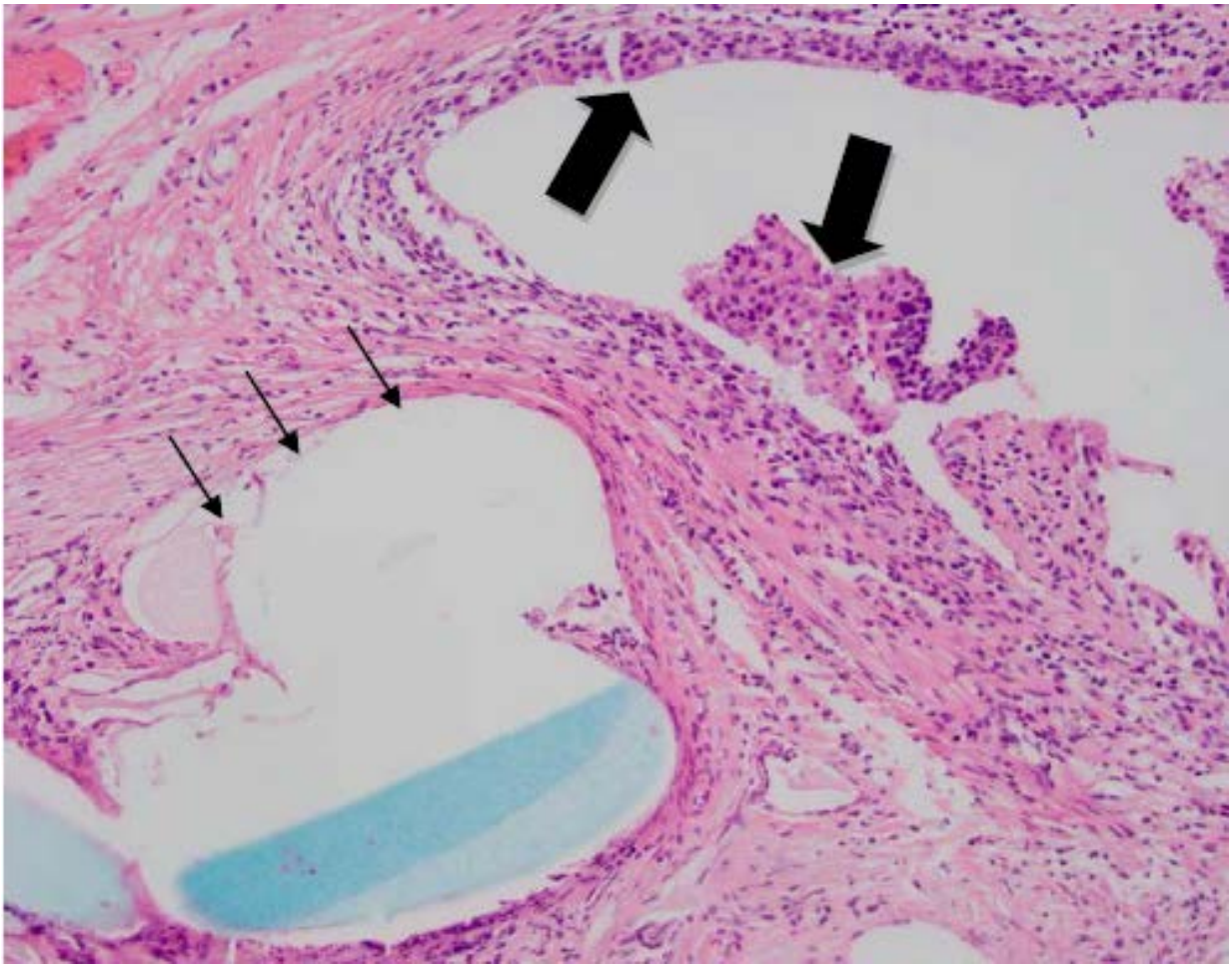

Figure 12: H&E of mesh explanted from Plaintiff "SC" showing mesh filaments (thin arrows) with associated perifilamentous fibrosis and chronic inflammation, eroding through the urothelial lining (block arrows) (200x magnification).

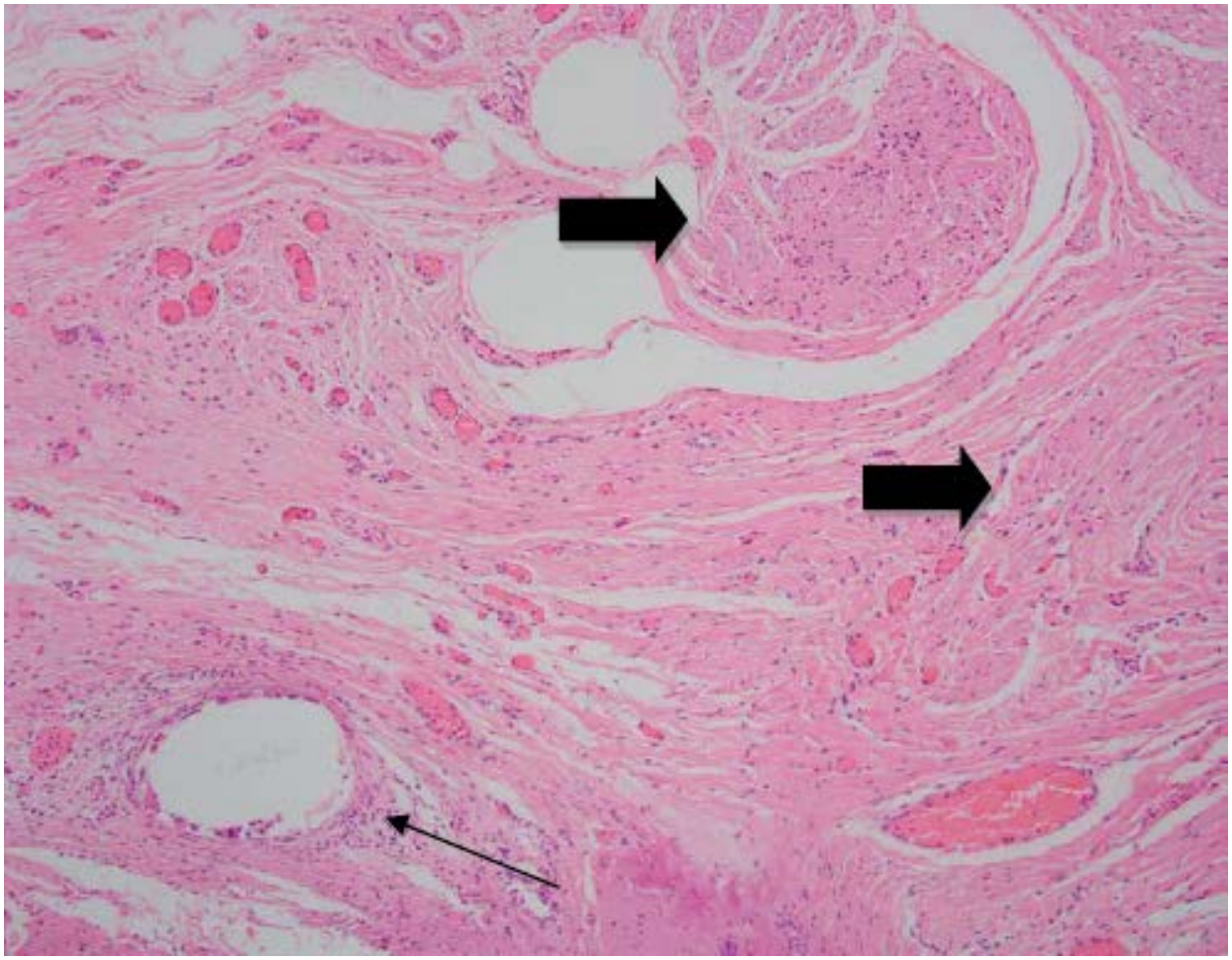

Figure 13: Histological section of mesh explanted from Plaintiff "SC" showing mesh filament (thin arrow) with associated perifilamentous fibrosis and chronic inflammation, involving the bladder smooth muscle bundles (block arrows) (100x magnification).

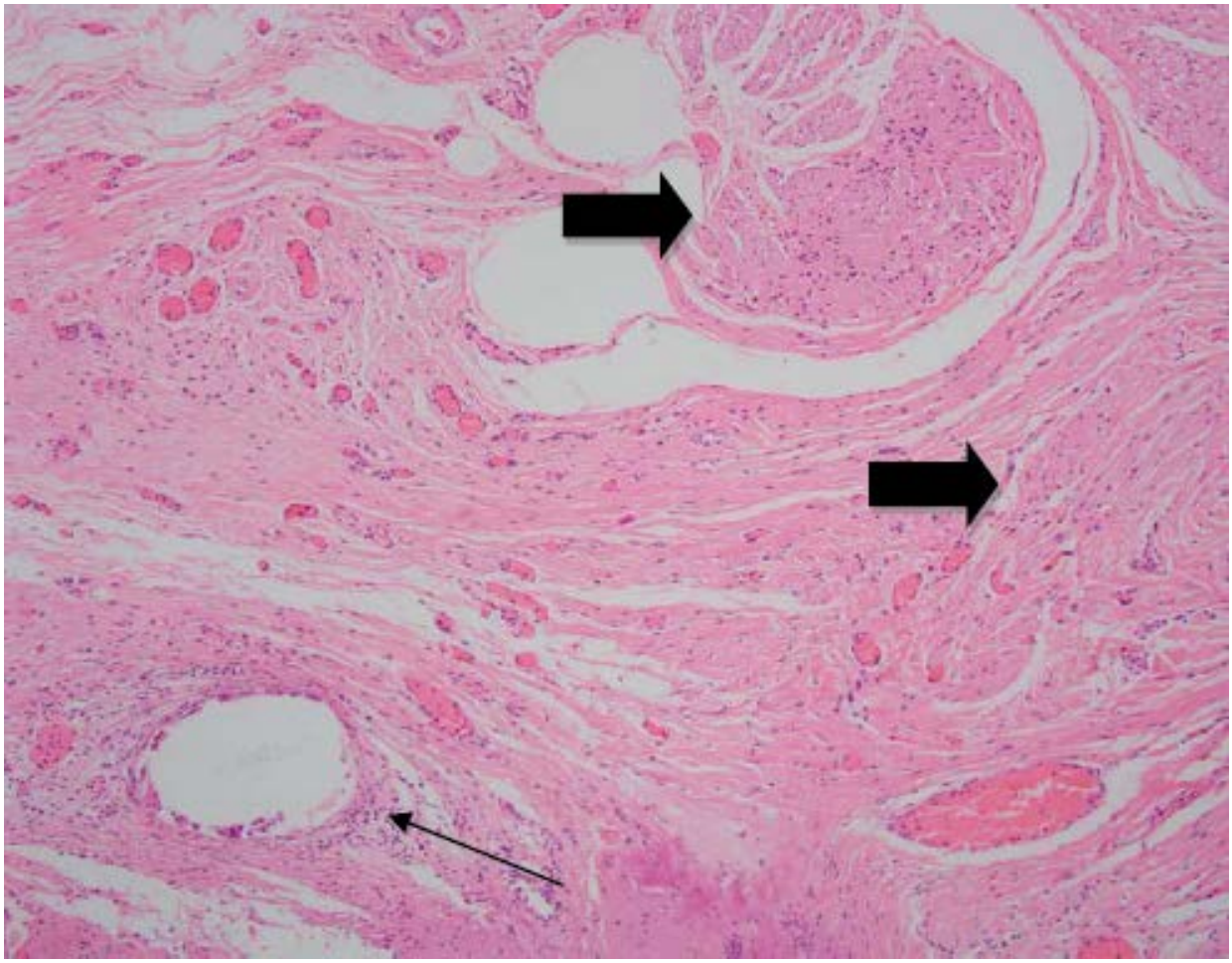

Figure 14: H&E of mesh explanted from Plaintiff "SC" showing mesh filament (thin arrow) with associated perifilamentous fibrosis and chronic inflammation, involving the bladder smooth muscle bundles (block arrows) (100x magnification).

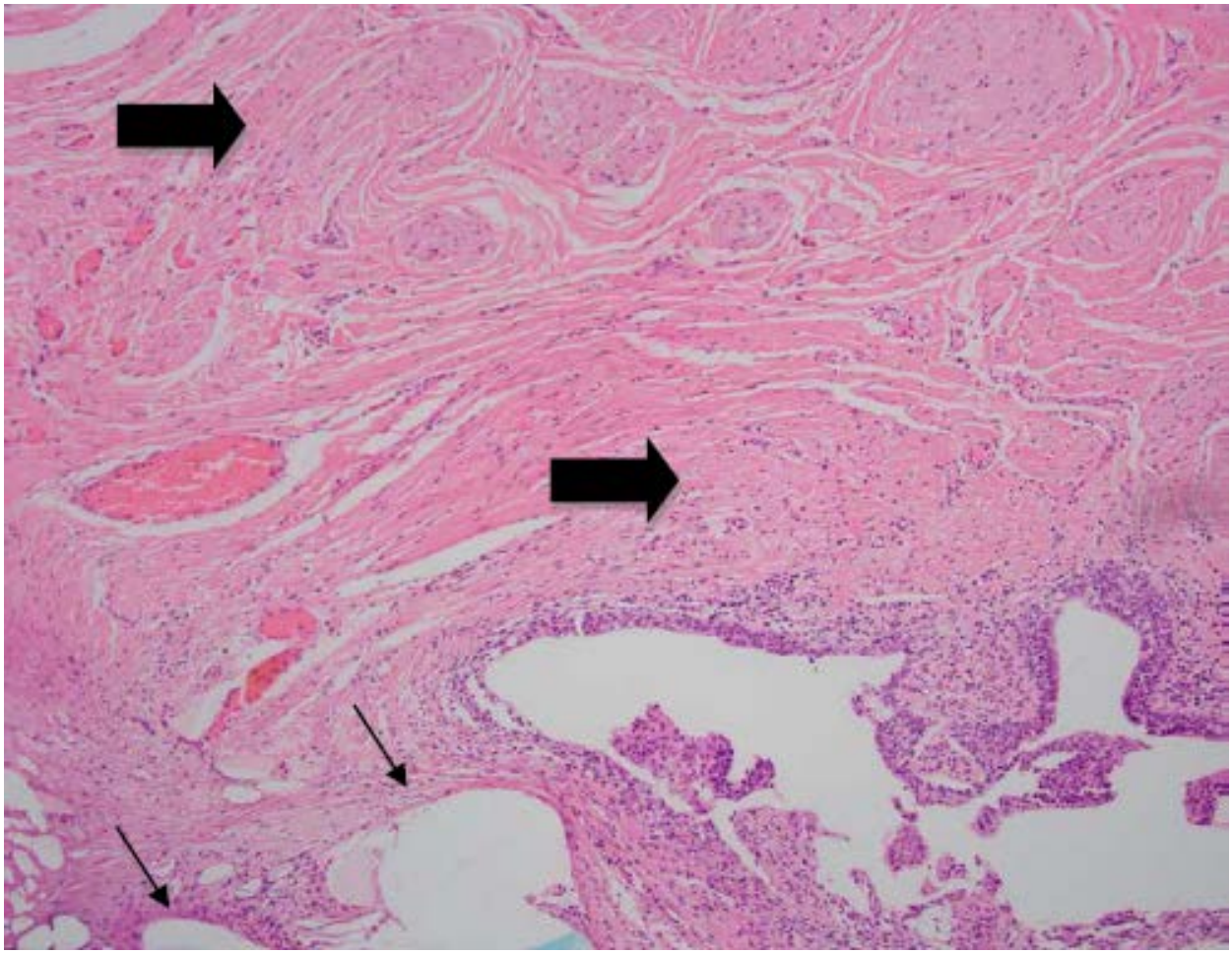

Figure 15: Another histology section of mesh explanted from Plaintiff "SC" showing mesh filaments (thin arrows) eroding into the urothelium with associated perifilamentous fibrosis and chronic inflammation, involving the bladder smooth muscle bundles (block arrows) (100x magnification).

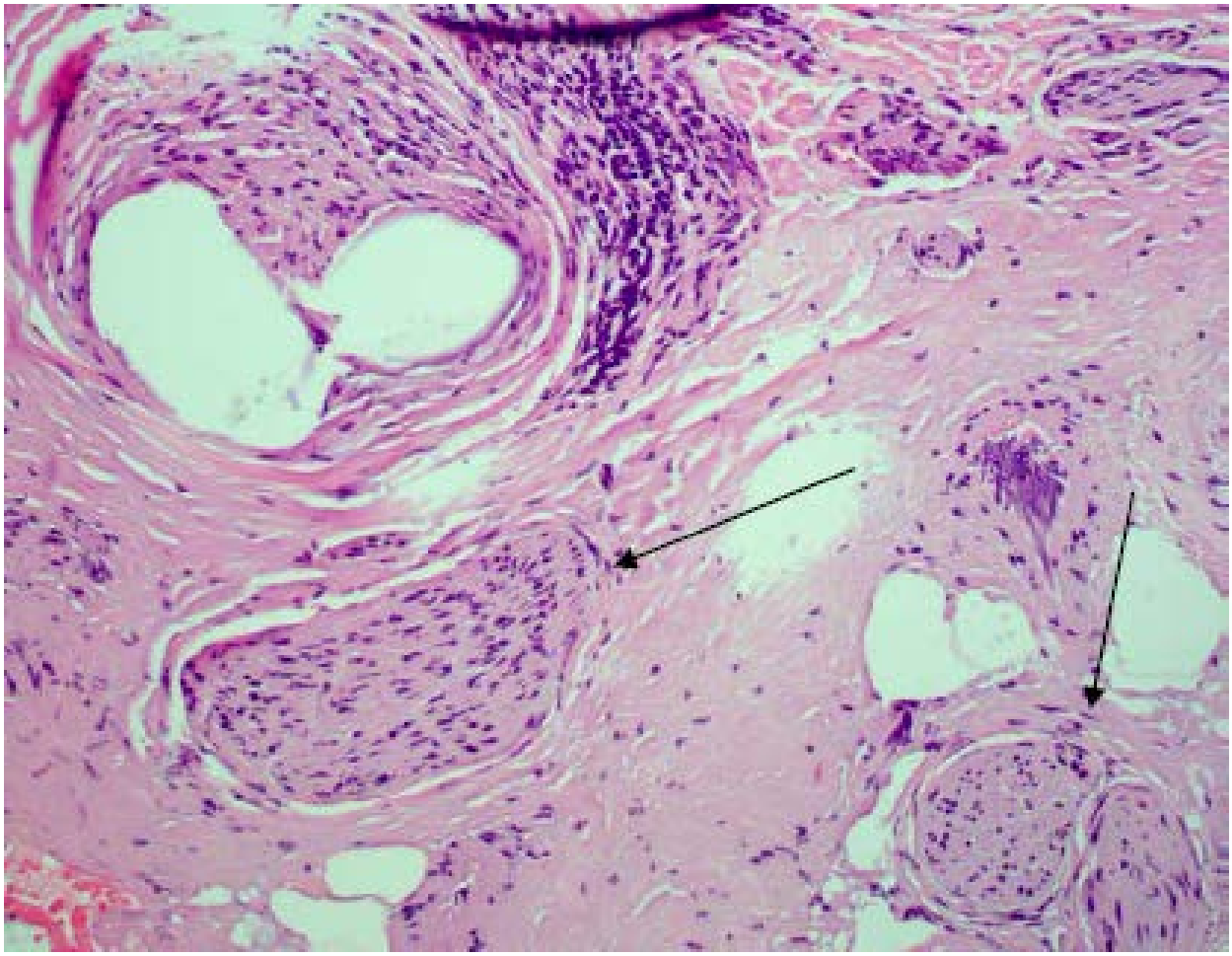

Figure 16: H&E stain of mesh specimen explanted from Plaintiff "MM" in which the prominence of the nerves (arrows) is evident without immunohistochemical stains, each surrounded by scar fibrosis (200x magnification).

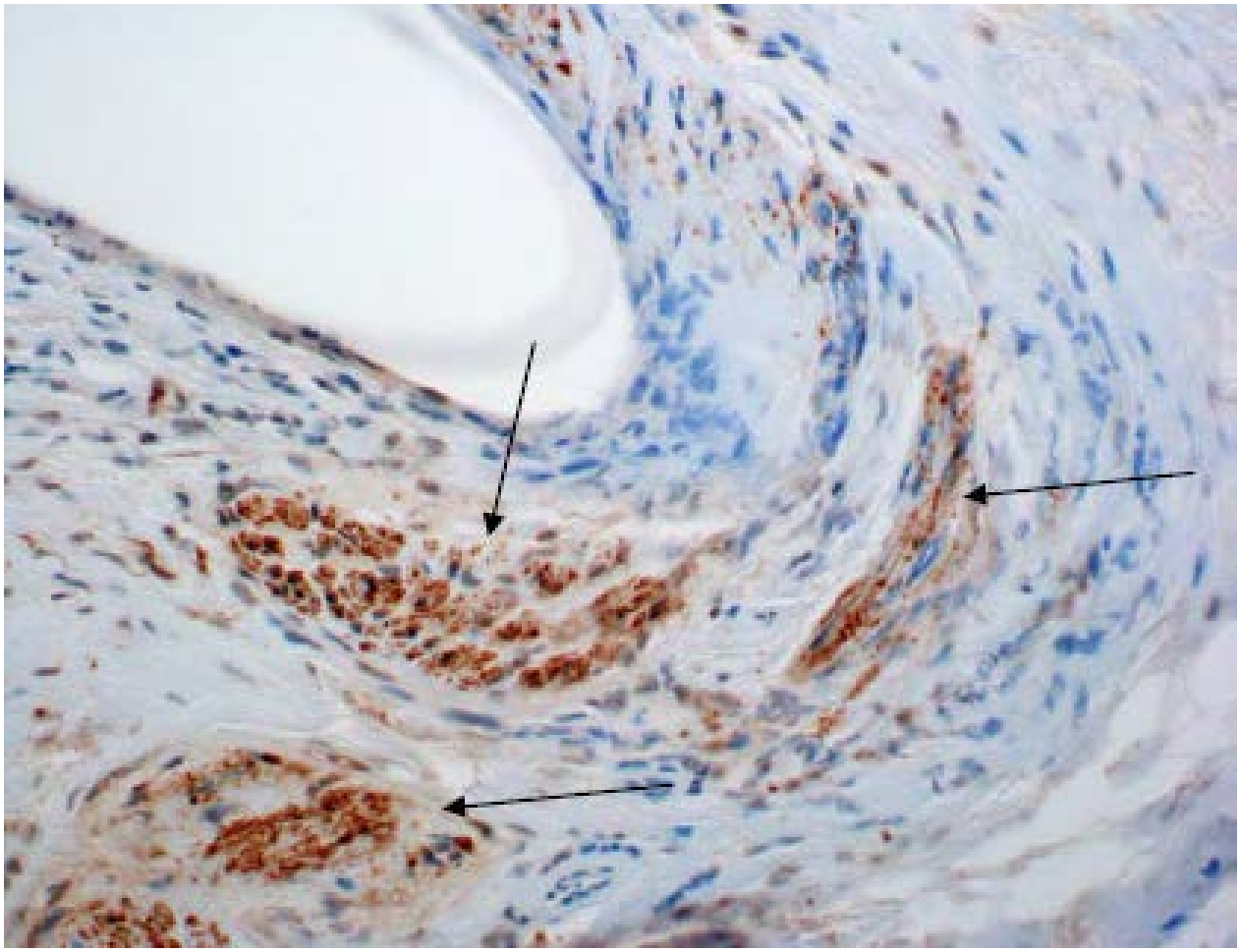

Figure 17: S100 stain of mesh explanted from Plaintiff "MM" highlighting the numerous, brown-staining nerves (arrow) that surround a large mesh filament and appear distorted and compressed by the associated inflammatory and fibrous response (400x magnification).

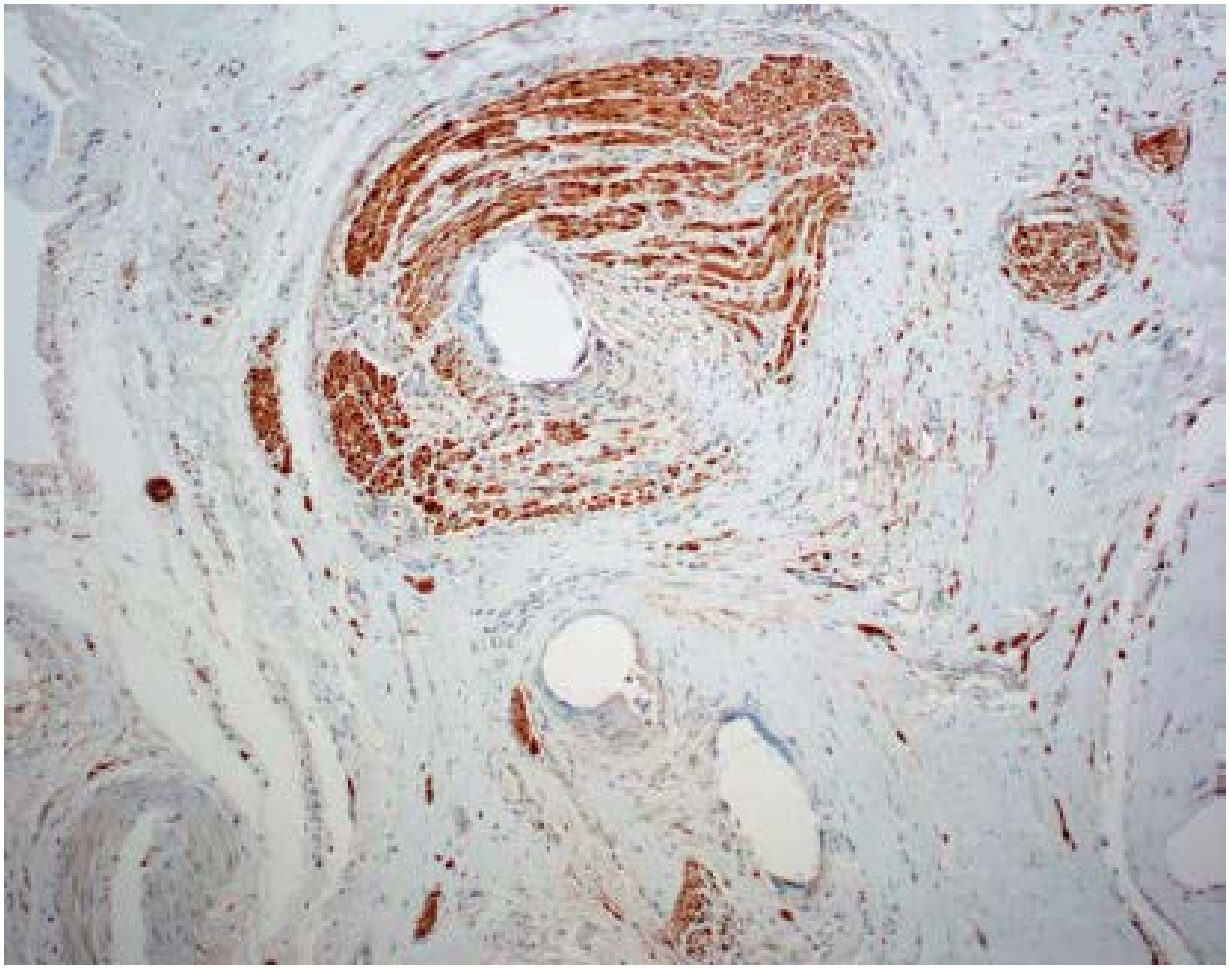

Figure 18: An S100 stain of mesh specimen explanted from Plaintiff "MM" highlighting the prominent and hyperplastic neural proliferation associated with a mesh filament, consistent with a "traumatic neuroma" (100x magnification).

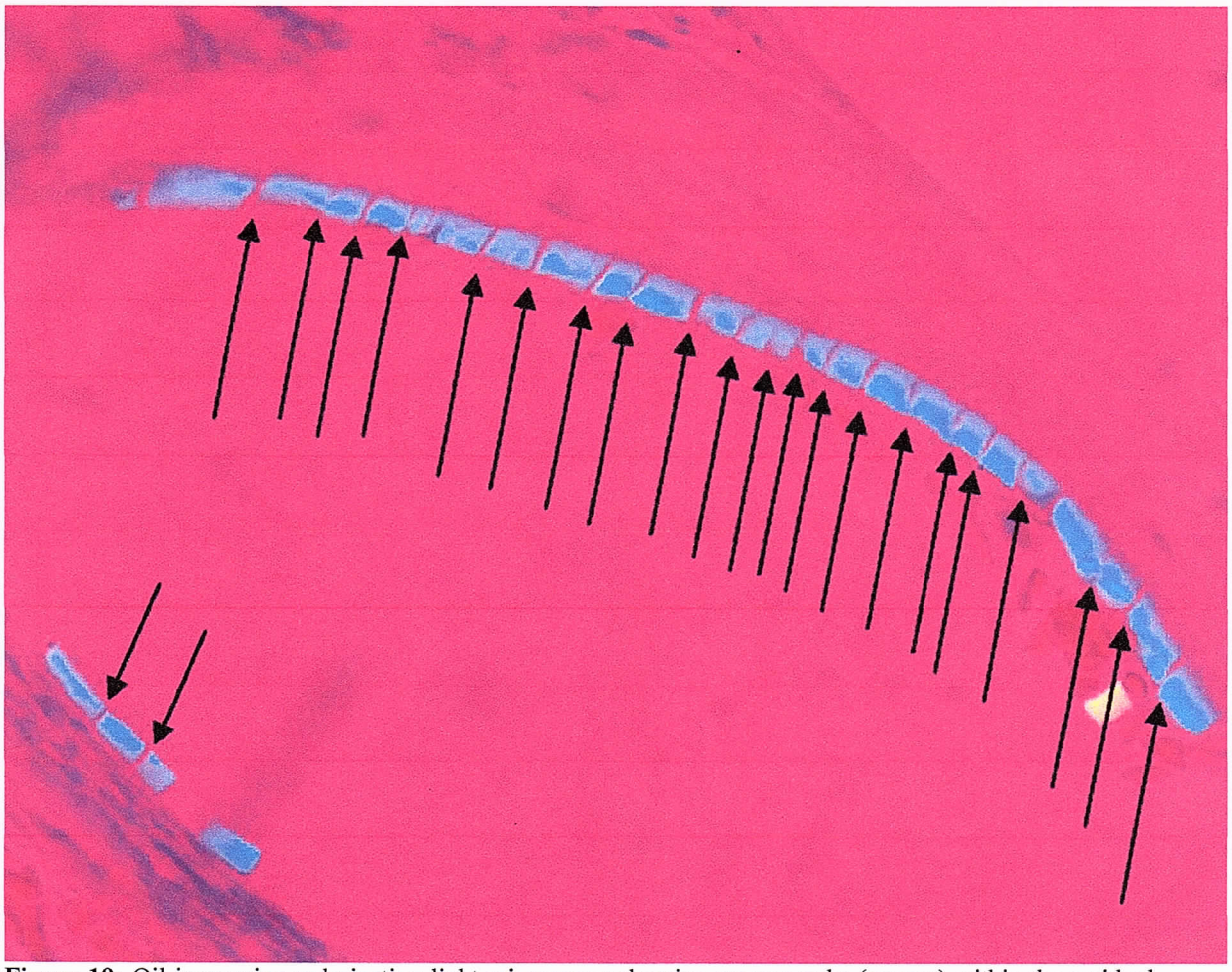

Figure 19: Oil immersion polarization light microscopy showing many cracks (arrows) within the residual polypropylene bark (1000x magnification) of mesh explanted from Plaintiff "TH".

7/1/16
DATE

Paul J. Michaels, M.D

EXHIBIT A

PAUL J. MICHAELS, M.D.

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PROFESSIONAL EXPERIENCE

February 2013 – Present

Pathologist at Clinical Pathology Associates Austin, TX

- Member of ~40 pathologist group that covers several major hospitals in the Central Texas region (including the Austin area and surrounding communities, San Marcos, and San Antonio)
- Anatomic pathology responsibilities include frozen sections, sign-out of complex surgical specimens, review of numerous outpatient biopsies, GYN and NON-GYN cytology, presentation at tumor board conferences, and performing hospital autopsies
- Clinical pathology responsibilities include peripheral blood smear and body fluid review, crystal identification, assessment of blood transfusion reactions and RBC antibody serology work-ups, TEG analysis, and evaluation of protein electrophoresis panels
- Director of the Fine Needle Aspiration Clinic at University Medical Center, Brackenridge (February 2013 – Present)
- Medical Director of North Austin CPL Stat Laboratory (April 2013 – Present)
- Medical Director of South Austin CPL Stat Laboratory (January 2014 – Present)
- College of American Pathologists (CAP) Inspections:
  - Team Leader (June 2014)

July 2006 – January 2013

Pathologist at Laboratory Medicine Consultants/Aurora Diagnostics Las Vegas, NV

- Member of a ~18 pathologist group that covered several hospitals in the southern Nevada and northwestern Arizona region
- Anatomic pathology responsibilities include frozen sections, sign-out of complex surgical specimens, review of numerous outpatient biopsies, GYN and NON-GYN cytology, coverage of outpatient FNA clinic, presentation at numerous tumor board conferences, and performing hospital autopsies
- Clinical pathology responsibilities include peripheral blood smear and body fluid review, crystal identification, assessment of blood transfusion reactions, protein electrophoresis and immunofixation analysis, and interpretation of various chemistry, lipid, coagulation, and serologic laboratory test panels
- Medical Director of MountainView Hospital Laboratory (January 2010 – January 2013):
  - Oversight of daily laboratory operations (~270 bed hospital)
  - Annual review of all laboratory policies
  - Member of the hospital Medical Executive Committee and Quality Council
- Laboratory Director of various ambulatory surgery centers, including:
  - Durango Surgery Center (February 2009 – January 2013)
  - Tenaya Surgery Center (January 2009 – January 2013)
  - Stonecreek Surgery Center (June 2008 – January 2013)
  - Las Vegas Regional Surgery Center (June 2007 – December 2007)
- Cytopathology Laboratory Director (August 2007 – January 2010)
  - Provided daily oversight of our centralized cytology laboratory for over 1,000 inpatient beds and numerous outpatient clinics in the surrounding community
  - Reviewed quarterly QA statistics, including NON-GYN/Surgical specimen correlation cases

- Cancer Conference Coordinator (January 2007 – December 2009) and Cancer Program Activity Coordinator (May 2007 – December 2009)
    - Reviewed all monthly cancer cases to assure compliance with Commission on Cancer standards
    - Provided oversight for all hospital Tumor Board conferences, including organization of the annual cancer conference assignments for all pathologists in the group
    - Attended bimonthly Sunrise Hospital Cancer Committee meetings
- College of American Pathologists (CAP) Inspections:
    - Team Leader (September 2009, August 2010)
    - Team Member (March 2007, September 2010)
- In 2012, was voted a "Top Doctor" in Pathology in Las Vegas, NV by Consumers' Checkbook of Washington, D.C., published in *Vegas Seven* magazine (2/23/2012).
    - Received the most mentions of any pathologist in the city

October 2004 – June 2006
Locum Tenens Pathologist at North Shore Pathologists Salem, MA

- Independently performed autopsies that occurred during the weekends and holidays
- Prepared and signed-out (cosigned) the autopsy reports with an attending/supervising pathologist

EDUCATION/CLINICAL TRAINING

July 2005 – June 2006
Cytopathology Fellowship, Massachusetts General Hospital/Harvard Medical School

June 2001 – June 2005
Anatomic/Clinical Pathology Residency, Massachusetts General Hospital/Harvard Medical School

- Chief Resident, Anatomic Pathology (June 2004 – November 2004)
- Resident Representative for Mentoring, American Society of Cytopathology, Ethics and Conduct Committee (June 2004 – June 2006)

August 1996 – June 2001
Doctorate of Medicine, University of California, Los Angeles

- Post Sophomore Fellowship in Anatomic/Clinical Pathology, Combined UCLA/Cedars Sinai Program (June 1998 – June 1999)
- Alpha Omega Alpha Honor Society (Elected 2001)

September 1992 – September 1995
Bachelor of Science, University of California, Irvine

- Major in Biological Sciences with a Minor in Microbiology, *Cum Laude*
- Phi Beta Kappa Honor Society (Elected 1995)
- UC Regents Scholar (1992 – 1995)

ACADEMIC APPOINTMENTS

June 2009 – June 2013
Adjunct Associate Professor of Pathology, Touro University of Nevada, College of Osteopathic Medicine

June 2001 – June 2006
Clinical Fellow in Pathology, Massachusetts General Hospital/Harvard Medical School

MEDICAL LICENSURE AND CERTIFICATION

October 2006 – Present
American Board of Pathology, Cytopathology (Time Limited, Recertified - March, 2015)

August 2005 – Present
American Board of Pathology, Anatomic and Clinical Pathology (Time Unlimited)

December 2012 – Present
Texas Medical Board (License # P5108)

December 2012 – Present
California Medical Board (License # C55645)

August 2006 – Present
Utah Medical Board (License # 6228782-1205)

June 2006 – Present
Nevada Medical Board (License #11907)

June 2006 – Present
Arizona Medical Board (License #35644)

October 2004 – June 2007
Massachusetts Medical Board (License #223088)

PROFESSIONAL ORGANIZATIONS

Texas Medical Association (TMA)
Travis County Medical Society (TCMS)
Texas Society of Pathologists (TSP)
American Society of Cytopathology (ASC)
American Society of Clinical Pathology (ASCP)
College of American Pathologists (CAP)
United States and Canadian Academy of Pathology (USCAP)

ACADEMIC PUBLICATIONS

Pusztaszeri M. Wang H, Cibas ES, Powers CN, Bongiovanni M, Ali S, Khurana KK, **Michaels PJ**, and Faquin WC. Fine-needle Aspiration Biopsy of Secondary Neoplasms of the Thyroid Gland: a Multi-institutional Study of 62 Cases. *Cancer Cytopathol* 2015;123:19-29.

Lewis Jr. BA, Zebrowski B, Yumiaco NS, **Michaels P**, and Erling M. Case Report of Paratesticular Liposarcoma with Metachronous Large Renal Cell Carcinoma. *Curr Urol* 2010;4:162-163.

Pitman MB, **Michaels PJ**, Deshpande V, Brugge WR, and Bounds BC. Cytological and Cyst Fluid Analysis of Small (<3 cm) Branch Duct Intraductal Papillary Mucinous Neoplasms Adds Value to Patient Management Decisions. *Pancreatology* 2008;8:277-84.

Michaels PJ, Brachtel EF, Bounds BC, Brugge WR, and Pitman MB. Intraductal Papillary Mucinous Neoplasms (IPMN) of the Pancreas: Cytologic Analysis and Correlation with Histologic Grade. *Cancer* 2006;108:163-73.

Steele DJ and **Michaels PJ**. Case Records of the Massachusetts General Hospital. Weekly Clinicopahtological Exercises. Case 40-2004- A 42-year-old Woman with Long-Standing Hematuria. *N Engl J Med* 2004;351:2851-9.

Michaels PJ, Espejo ML, Kobashigawa J, Alejos JC, Burch C, Takemoto S, Reed EF, and Fishbein MC. Humoral Rejection in Cardiac Transplantation: Risk Factors, Hemodynamic

Consequences and Relationship to Transplant Coronary Artery Disease. *J Heart Lung Transpl* 2003;1:58-69.

Michaels PJ, Fishbein MC, and Colvin RB. Humoral Rejection in Human Transplantation. *Springer Semin Immunopathol* 2003;25:119-140.

Marchevsky AM, Lau SK, Khanafshar I, Ockhart C, Phan A, **Michaels PJ**, and Fishbein MC. Internet Teleconferencing Method for Telepathology Consultations from Lung and Heart Transplant Patients. *Hum Pathol* 2002;33:410-4.

Michaels PJ, Kobashigawa J, Laks H, Azarbal A, Espejo ML, Chen L, and Fishbein MC. Differential Expression of RANTES Chemokine, TGF-β, and Leukocyte Phenotype in Acute Cellular Rejection and Quilty B Lesions. *J Heart Lung Transpl* 2000;20:407-16.

Michaels PJ, Kobashigawa J, Child JS, and Fishbein MC. Chronic Right Sided Myocarditis Mimicking Arrhythmogenic Right Ventricular Dysplasia. *Hum Pathol* 2000;31:618-21.

Marchevsky A, Lockhart C, Phan A, **Michaels PJ**, and Fishbein MC. Web-Based Teleconferencing Techniques as Inexpensive Tools for Transplant Patients. *Lab Invest* 2000;80:37A.

Michaels PJ and Mautz WJ. Effects of Inhaled Ozone and Formaldehyde on Tracheal Epithelial Secretion of Rats Exposed During Rest and Exercise. *Journal of Undergraduate Research in the Biological Sciences* 1995;25:779-90.

PRESENTATIONS

INVITED TALKS:

April 2015

"The Surgical Pathology of Dysphonia" for the Masters Program in Speech and Language Pathology at University of the Pacific, Stockton, California.

January 2014

"Cytology From Sin City" at University of Colorado, Denver, Department of Pathology and Laboratory Medicine. Invited Grand Rounds Speaker.
"Cytology Jeopardy" at University of Colorado, Denver, Department of Pathology and Laboratory Medicine. Invited Unknown Conference for Residents.

March 2013

"Confounding Metastatic Breast Cancer Controversy" presented at the 23rd Annual National Interdisciplinary Breast Center Conference for the National Consortium of Breast Centers. Planet Hollywood Resort & Casino. Las Vegas, Nevada.

March 2012

Panelist for *"Interesting Cases: What Would You Have Done?"* presented at the 22nd Annual National Interdisciplinary Breast Center Conference for the National Consortium of Breast Centers. Paris Las Vegas Hotel & Casino. Las Vegas, Nevada.

November 2010

"Cytology From Sin City 2" at Massachusetts General Hospital and Brigham and Women's Hospital, Departments of Pathology, Harvard Medical School.

February 2009

"Cytology From Sin City" at Massachusetts General Hospital, Brigham and Women's Hospital, and Beth Israel Deaconess, Departments of Pathology, Harvard Medical School.

December 2003

"Thin Basement Membrane Nephropathy and Alport Syndrome" at Massachusetts General Hospital, Clinicopathologic Conference (Published in *N Engl J Med*), Harvard Medical School.

PLATFORM PRESENTATIONS:

Michaels PJ, Brachtel EF, Bounds BC, Brugge WR, and Pitman MB. Intraductal Papillary Mucinous Neoplasm (IPMN) of the Pancreas: Cytologic Analysis and Correlation with Histologic Grade. Annual Meeting of United States and Canadian Academy of Pathology. March 2004. Vancouver, British Columbia, Canada.

Michaels PJ, Kobashigawa J, Laks H, Azarbal A, Espejo ML, Chen L, and Fishbein MC. Differential Expression of RANTES Chemokine and Leukocyte Phenotype in Acute Cellular Rejection and Quilty B Lesions. 20th International Society of Heart and Lung Transplantation Annual Meeting. April, 2000. Osaka, Japan.

Kakkis JL, **Michaels PJ**, Ma JP, Ke B, Kupiec-Weglinski J, Imagawa DK, and Busuttil RW. Pravastatin Prolongs Rat Survival after Orthotopic Liver Transplantation by Decreasing the Expression of β2-Glycoprotein-1 and Proinflammatory Cytokines. World Congress of the Transplantation Society. July 12-17, 1998. Montreal, Quebec, Canada.

POSTER PRESENTATIONS:

Michaels PJ, Bounds BC, Brugge WR, Lewandrowski K, Pitman MB. The Clinical Utility of Cyst Fluid Analysis in Conjunction with Cytological Evaluation in the Preoperative Characterization and Subclassification of Pancreatic Mucinous Cysts. 52nd American Society of Cytopathology Annual Meeting. November, 2004. Chicago, IL.

Michaels PJ, Brachtel EF, Bounds BC, Brugge WR, and Pitman MB. Intraductal Papillary Mucinous Neoplams (IPMN) of the Pancreas: Cytologic Analysis and Correlation with Histologic Grade. Massachusetts General Hospital Clinical Research Day. June, 2004. Boston, MA.

Michaels PJ, Kobashigawa J, Espejo ML, Alejos JC, Burch C, and Fishbein MC. Humoral Rejection in Cardiac Transplantation: Recent UCLA Experience. Sixth Banff Conference on Allograft Pathology. April, 2001. Banff, Canada.

Marchevsky A, Lockhart C, Phan A, **Michaels PJ**, and Fishbein MC. Web-based Teleconferencing Techniques as Inexpensive Tools for Transplant Patients. 2000 Annual Meeting of United States and Canadian Academy of Pathology. March, 2000. New Orleans, LA.

Kakkis JL, Schmit P, **Michaels PJ**, and Thompson J. Management of Gallstone Disease During Pregnancy in the Era of Laparoscopic Cholecystectomy. The Southwestern Surgical Congress. April, 1999. Coronado, CA.

Kakkis JL, **Michaels PJ**, Ke B, Zhao D, Kato H, Imagawa D, Kupiec-Weglinski JW, and Busuttil RW. Treatment with Pravastatin Ameliorates Rejection and Improves Survival in Liver Transplanted Rats. International Congress on Immunosuppression. December 1998. Orlando, FL.

Kakkis JL, **Michaels PJ**, Gornbein J, Terasaki P, Imagawa D, Busuttil R. Multivariate Analysis of Risk Factors in 1,008 Orthotopic Liver Transplant Recipients Reveals Significant Influence of Panel Reactive Antibody on Patient and Graft Survival. Annual Meeting of the American College of Surgeons, October 1998. Orlando, FL.

Kakkis JL, **Michaels PJ**, Ma JP, Ke B, Kupiec-Weglinski J, Imagawa DK, and Busuttil RW. Pravastatin-induced Survival in Rat Orthotopic Liver Transplantation is Accompanied by Diminished Expression of β2-Glycoprotein-1 and Proinflammatory Cytokines. American Society of Transplant Physicians. May, 1998. Chicago, IL.

Michaels PJ, Ma J, Zhao D, Imagawa D, Busuttil R, and Kakkis JL. Pravastatin Treatment is Associated with Downregulation of TGF-β and TNF-α in Liver Transplanted Rats. 1997 Short Term Training Program Poster Session. Los Angeles, CA.

Kakkis JL, **Michaels PJ**, Ma JP, Ke B, Zhao D, Imagawa DK, and Busuttil RW. Analysis of Genetic Modifications in Liver Transplanted Rats Utilizing Messenger RNA Differential display. American Society of Transplant Surgeons. May, 1997. Chicago, IL.

EXHIBIT B

PAUL J. MICHAELS, M.D.

4214 Speedway, Austin, Texas 78751
PHONE: (512) 808-6711 EMAIL: pauljmichaels@gmail.com

FOUR-YEAR PRIOR EXPERT TESTIMONIES

DEPOSTIONS	**DATE**
Elizabeth Dees (Plaintiff) vs. Wyeth - *Deposed as expert Pathology witness for Plaintiff*	3/16/13
Kathryn Willis (Plaintiff) vs. Wyeth - *Deposed as expert Pathology witness for Plaintiff*	3/16/13
Nikolina Bundalo (Plaintiff) vs. Jozsef Zority, M.D. - *Deposed as expert Pathology witness for Plaintiff*	4/13/13
Mike Bendfeldt and Betty Muhr-Bendfeldt (Plaintiff) vs. HRC Medical Centers, INC. - *Deposed as expert Pathology witness for Plaintiff*	4/19/16
In Re: Ethicon, Inc., Pelvic Repair System Products Liability Litigation - *Deposed as general Pathology expert witness for Plaintiffs*	6/18/16
Tamara Carter vs. Ethicon, Inc., et al. - *Deposed as expert Pathology witness for Plaintiff*	6/18/16
Sandra Childress vs. Ethicon, Inc., et al. - *Deposed as expert Pathology witness for Plaintiff*	6/18/16
Marion Chrysler vs. Ethicon, Inc., et al. - *Deposed as expert Pathology witness for Plaintiff*	6/18/16
Melissa Sanders vs. Ethicon, Inc., et al. - *Deposed as expert Pathology witness for Plaintiff*	6/19/16
Ana Sierra vs. Ethicon, Inc., et al. - *Deposed as expert Pathology witness for Plaintiff*	6/19/16
Toni Hernandez vs. Ethicon, Inc., et al. - *Deposed as expert Pathology witness for Plaintiff*	6/19/16

COURT TESTIMONIES

Toshiko Okuda (Plaintiff) vs. Wyeth 8/20/12
- *Testified as expert Pathology witness for Plaintiff*

EXHIBIT C

PAUL J. MICHAELS, M.D.

4214 Speedway, Austin, TX 78751
PHONE: (512) 808-6711 EMAIL: pauljmichaels@gmail.com

SCHEDULE OF FEES

1.	Cognitive work, lab work, records review, meetings	$500.00/hour
2.	Depositions*	$500.00/hour
3.	Court Testimony**	$3,000/half day $5,500/whole day
4.	Overnight	As above, plus per diem ($1000.00)

** Depositions are charged at a two hour minimum. Any cancellations less than 48 hours will be charged two hours. Paid by side taking the deposition. Due at the time of deposition. No exceptions*

*** Cancellations of court appearances, etc., occurring less than 48 hours of the event will be billed at 50% of the scheduled rate*

EXHIBIT D


PAUL J. MICHAELS, M.D.

BOARD CERTIFIED IN ANATOMIC AND CLINICAL PATHOLOGY, AND CYTOPATHOLOGY


Reliance List

1. Abed H, Rahn DD, Lowenstein L, et al. Systematic Review Group of the Society of Gynecologic Surgeons. Incidence and management of graft erosion, wound granulation, and dyspareunia following vaginal prolapse repair with graft materials: a systematic review. *Int Urogynecol J* 2011;22:789-98.
2. Altman D, Vayrynen T, Engh ME, et a.; Nordic Transvaginal Mesh Group. Anterior colporrhaphy versus transvaginal mesh for pelvic-organ prolapse. *N Engl J Med* 2011;364:1826-36.
3. Amid PK. Classification of biomaterials and their related complications in abdominal wall hernia surgery. *Hernia* 1997;1:15-21.
4. Anderson JM, Rodriguez A, and Chang DT. Foreign body reaction to biomaterials. *Semin Immunol* 2008;20:86-100.
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6. Barone WR, Moalli PA, and Abramowitch SD. Textile properties of synthetic prolapse mesh in response to uniaxial loading. *Am J Obstet Gynecol* 2016 Mar 18.
7. Bendavid R, Lou W, Koch A, et al. Mesh-related SIN Syndrome. A surreptitious irreversible neuralgia and its morphologic background in the etiology of post-herniorrhaphy pain. *Int J Clin Med* 2014;5:799-810.
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10. Blavias JG, Purohit RS, Benedon MS, et al. Safety considerations for synthetic sling surgery. *Nat Rev Urol* 2015;12:481-509.
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17. Crosby EC, Abernethy M, Berger MB, et al. Symptom resolution after operative management of complications from transvaginal mesh. *Obstet Gynecol* 2014;123:134-9.
18. Dallenbach P. To mesh or not to mesh: a review of pelvic organ reconstructive surgery. *Int J Womens Health* 2015;7:331-43.
19. Danford JM, Osborn DJ, Reynolds WS, et al. Postoperative pain outcomes after transvaginal mesh revision. *Int Urogynecol J* 2015;26:65-69.
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21. de Tayrac R and Letouzey V. Basic science and clinical aspects of mesh infection in pelvic floor reconstructive surgery. *Int Urogynecol J* 2011;22:775-80.

22. Diwadkar GB, Barber MD, Feiner B, et al. Complication and reoperation rates after apical vaginal prolapse surgical repair: a systematic review. *Obstet Gynecol* 2009;113:367-73.
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32. Hill AJ, Unger CA, Solomon ER, et al. Histopathology of excised midurethral sling mesh. *Int Urogynecol J* 2015;26:591-5.
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61. Nieminen K, Hiltunen R, Takala T, et al. Outcomes after anterior vaginal wall repair with mesh: a randomized, controlled trial with a 3 year follow-up. *Am J Obstet Gynecol* 2010;203:235.e1-e8.
62. Nolfi AL, Brown BN, Liang R, et al. Host response to synthetic mesh in women with mesh complications. *Am J Obstet Gynecol* 2016 Apr 16.
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Ethicon-Related References

1. Arnaud deposition 9/25/13 772:25 to 777:16; 779:4-11
2. ETH.MESH.00870466: 2006 Expert Meeting Norderstedt
3. ETH.MESH.01782867: "Factors Related to Mesh Shrinkage" PowerPoint presentation by Kestin Spychaj
4. Hinoul deposition 4/15/12 99:09-99:25, 4/6/12 518:14-520:20, 6/26/13 175:1-176:17, 184:18-22 328:10-24
5. Owens deposition 9/12/2012 98:11 to 99:07
6. Batke deposition 08/01/13 87:12 to 88:10, 113:3 to 114:3, 257:23 to 259:13
7. Arnaud deposition 9/25/13 769:23 to 770:4
8. ETH.MESH.04037600: Innovations in mesh development
9. ETH.MESH.05920616: 7/20/07; Chomiak, Martin to Batke, Boris; Jamieson, Gillian; Koehler, Petra; Hellhammer, Dr. Brigitte SUBJECT: Defining light weight mesh
10. ETH.MESH.05585033
11. ETH.MESH.05446127: 3/13/2006 Holste, Dr. Joerg to Engel, Dr. Dieter; Manley, Quentin; Storch, Mark L. SUBJECT: AW: Mesh and Tissue Contraction in Animal
12. ETH.MESH.050475773
13. ETH.MESH.04015102: 3/01/12 Batke, Boris to Mayes, Casey SUBJECT: AW: AGES Pelvic Floor Conference-Gala Dinner Invitation
14. ETH.MESH.09651393: Invention Disclosure
15. ETH.MESH.05585066: "Ultrapro" PowerPoint presentation by Boris Batke
16. ETH.MESH.05916450: "Chronic Pain Prevention/future – Bioengineer's point of view"
17. ETH.MESH.00237968: "R&D Perspective – The Journey from Prolift to Prolift +M" PowerPoint presentation by Cliff Volpe
18. ETH.MESH.05479411: Heavyweight to Lightweight Meshes PowerPoint
19. Holste deposition 07/29/13 51:3 to 53:6, 55:22 to 57:4
20. Vailhe deposition 06/20/13 182:2 to 185:5
21. ETH.MESH.01774758: December 2006 email regarding TVM Group mesh design input
22. ETH.MESH.02992139: Lightning Clinical Strategy dated 11/22/06
23. ETH.MESH.05447475: Email from Dieter Engel to John Gillespie et al. re: How inert is polypropylene?
24. ETH.MESH.02227224: PowerPoint presentation dated 05/09/08 titled MGPP Thunder Decision Meeting
25. ETH.MESH.00869908: Thunder Meeting Minutes dated 8/14/07
26. ETH.MESH.09557798: 7 Year Dog Study

27. ETH.MESH.02010834 – ETH.MESH.0201854: February 16, 2011 report written by Jurgen Trzewik and Christope Vailhe titled "Biomechanical consideration for Pelvic floor mesh design"
28. ETH.MESH.00033325: Professional Education PowerPoint presentation titled "The Science of Augmented Extracorporeal Reconstructive Pelvic Surgery" in which the "Ideal Mesh" is described.
29. ETH-65881: Gynecare Prolift IFU
30. ETH-00255: Ethicon Gynemesh PS 2006 marketing brochure
31. Scott Ciarrocca testimony 2012-03-29 00, (Page 340:9 to 340:12)
32. Aaron Kirkemo testimony 105:14-108:16
33. ETH.MESH.03924887: Meshes in Pelvic Floor Repair
34. Hinoul trial testimony *Gross, et al. vs Gynecare, et al* 01/16/13:1151:5-1156:14; 1087:1-17;1058:22-1059:3
35. ETH.MESH.00006636: Klosterhalfen B., Interim Report Mesh Explants Pelvic Floor Repair
36. ETH.MESH.02157879-02757880: Klosterhalfen B., Intermediate Report – Prolapse Mesh Explants 6/2009